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ARTICLE

The Relationship between Diet and Porous Cranial Lesions in the Southwest United States: A Review

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Abstract
Bioarchaeologists commonly record porous cranial lesions (PCLs). They have varied etiologies but are frequently associated with nutritional anemia without a differential diagnosis. This article provides a literature review, evaluates diet in the US Southwest over time, and identifies issues with associating PCLs with poor diet in this region. Generally, diet was adequate across time and space. Although maize was a dietary staple, other food items such as rabbits and amaranth provided complementary micronutrients. PCLs exhibit varied morphologies, which generally correspond with age: those characterized by fine, scattered porosity are associated with younger ages at death. Variation in PCL morphology indicates different and sometimes unrelated etiologies. Nutritional anemia is an insufficient explanation for PCL frequency in the Southwest, partly because the diet was adequate across time.

Resumen
Las lesiones craneales porosas (LCP) son se encuentran comúnmente en análisis bioarqueologicos. LCP tienen variedad de etiologías, pero frecuentemente se asocian con anemia nutricional sin diagnóstico diferencial. Este artículo propone una revisión de la literatura, evaluación la dieta en el suroeste de los Estados Unidos a través del tiempo e identifica problemas con la asociación de las LCP con mala alimentación en esta región. En general, la dieta fue adecuada a través del tiempo y región. Aunque el maíz era considerado como un alimento básico, otros alimentos (por ejemplo, conejos y amaranto) proporcionaban micronutrientes complementarios. Las LCP exhiben variedad en morfologías, que generalmente corresponden con la edad. Las LCP, caracterizadas por una porosidad fina y dispersa, se asocian con la mortalidad en poblaciones jóvenes. La variación en la morfología de LCP indica etiologías diferentes y a veces sin relación. La anemia nutricional es insuficiente para justificar la frecuencia de LCP en el suroeste americano, en parte porque la dieta fue apropiada a lo través del tiempo.

Keywords: anemia; stress indicators; iron deficiency; Southwest; cribra orbitalia; porotic hyperostosis

Palabras clave: anemia; indicadores de estrés; deficiencia de hierro; suroeste americano; cribra orbitalia; hiperostosis porótica

Skeletal lesions used as indicators of environmental stress in archaeological contexts are often etiologically complex and may be generated through multiple biological pathways. Cribra orbitalia (CO) and porotic hyperostosis (PH) are descriptive terms for porous cranial lesions (PCLs) found on the orbit roofs and cranial vault that cause pitting, porosity, or thickening of the affected bones (Brickley 2018; Lewis 2017:194). Often, they are assumed to have the same etiology: acquired anemia from nutritional deficiencies. The association of PCLs with iron-deficiency anemia in past peoples is attributable to Moseley (1965:141), although he stipulated that "it does not seem necessary . . . to ascribe the same etiology to

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all." Despite that caveat, PCLs, regardless of morphology, are consistently attributed to nutritional anemia without differential diagnosis (for more discussion, see Grauer 2019; Mays 2012; Ortner 2012). According to Ortner (2012:251), this association "has resulted in abnormalities being diagnosed as anemia when other diagnostic options are more probable. It also means that much of the literature on the prevalence of anemia . . . is likely to overestimate the true prevalence of the skeletal disorder in antiquity."

Failure to consider the complex biocultural processes that contribute to lesion formation may impede efforts to assess environmental impacts on patterns of morbidity and mortality in archaeological contexts. This article leverages data from an Ancestral Pueblo archaeological assemblage (AD 1100–1400s) and dietary data from a literature review to build on previous research examining relationships among PCLs, diet, and illness (Brickley et al. 2020; Cole and Waldron 2019; Mays 2012; Rivera and Mirazón Lahr 2017; Walker et al. 2009; Wapler et al. 2004).

Much like PCLs, anemia develops in varied conditions, including trauma, infection, renal failure, cancer, and metabolic disorders (Brickley et al. 2020; Ives 2018; Schattmann et al. 2016). This complexity may confound efforts to identify potential environmental factors such as diet and disease that contribute to PCL development. Additionally, studies suggest that CO and PH may have different etiologies (Rivera and Mirazón Lahr 2017; Schultz 2003; Walker et al. 2009), even when observed in the same person, because individuals can have comorbidities (see, e.g., Ives 2018). Recent work indicates different developmental trajectories for CO and PH, which may speak to their varied causes (O'Donnell et al. 2023). Because different biological pathways promoting PCL formation may involve different life-history trade-offs—for example, immune function versus growth (see Stearns 1992)—and associated costs such as constrained growth and elevated morbidity or mortality risks relevant to archaeological research questions, care should be taken in their interpretation.

This article reviews the literature and provides limited analyses to examine the association between PCLs and diet in the US Southwest. We examine bone changes in anemia and the relationship between anemia and PCLs in the Ancestral Pueblo world.

Background

Anemia and PCLs

Anemia occurs when red blood cells are low in number or dysfunctional, or hemoglobin concentration is lower than normal (Beutler 1988; WHO 2019a). Anemia has many potential causes and manifestations (Grauer 2019), and its presence signals an underlying health condition, but anemia itself is not a disease (Beutler 1988; Shine 1997). See Supplemental Table 1 for terminology and definitions.

PCLs are commonly attributed to acquired anemias, including iron-deficiency anemia (El-Najjar et al. 1975; Fink 1985; Hens et al. 2019; for discussion, see Brickley and Morgan 2023:343–344). Iron-deficiency anemia and anemia of inflammation (AoI) are the most often observed acquired anemias in clinical settings today (Ganz 2019). When AoI develops, it is often through iron sequestration or iron withholding (Spivak 2002), adaptive features of the immune system (Jurado 1997; Zucker et al. 1974).

AoI differs from iron deficiency because it typically does not deplete the body's total iron stores. Instead, serum ferritin—reflective of iron stores—is increased in AoI but decreased in iron-deficiency anemia (Nayak et al. 2018). Despite these differences, it is sometimes difficult for clinicians to differentiate between the two conditions (Schapkaitz et al. 2015), and they often co-occur (Ganz 2019). PCLs similar to those seen in congenital anemias are sometimes reported in individuals with iron-deficiency anemia (Eng 1958; Sheldon 1936), but they are rare compared to postcranial alterations in iron-deficiency anemia (Agarwal et al. 1970).

Vitamin B_{12} deficiency, which can cause megaloblastic anemia, has been suggested as a cause of PCLs (Martinson 2002; Walker et al. 2009). However, B_{12} deficiency in childhood, during which PCLs develop, is extremely rare (Lewis 2017; Mtvarelidze et al. 2009). This alone is inconsistent with the frequency of PCLs in the archaeological record. B_{12} deficiency is not definitively linked with hyperplastic skeletal lesions in the medical literature, although there is some evidence of a relationship to bone loss (Stone et al. 2004); for discussion, see Oxenham and Cavill (2010) and McIlvaine (2015). A recent article by Brickley (2024) provides an extended discussion and critique of the association of anemia with PCLs.

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Although PCL presence alone only serves as a generalized indicator of stress, PCL morphology is likely indicative of its etiology (e.g., Brickley et al. 2020:7; Mays 2018a; Ortner 2003). In congenital anemias, PCLs are often characterized by marrow hyperplasia and pitting/porosity (Chaichun et al. 2021; Cooley and Lee 1925). In infection, PCLs are often superficial to normal bone and not associated with expansion of the marrow space (Grauer 2019:520). Inflammation, which results from immune responses to many conditions, may cause lesions characterized by bone loss (Epsley et al. 2020). Nutrient deficiency can cause superficial new bone and microporosity (scurvy; Ortner 2003; Snoddy et al. 2018) or spiculation of bone surfaces and larger pores (rickets; Mays 2008:185, 2018b).

Developmental Factors Contributing to Lesion Formation

When observed in adults, PCLs are thought to represent developmental stress that an individual survived (Stuart-Macadam 1985), potentially signaling the initiation of critical physiological trade-offs that influence later morbidity and mortality risks (McPherson 2021; Temple 2019). Recent work indicates that the developmental window for CO closes around eight years of age, but PH may continue to form in later life-history phases (O'Donnell et al. 2023). Limited evidence suggests that pediatric males are more likely to develop CO than females (O'Donnell et al. 2022; Sheridan and Van Gerven 1997). There are also indications that different PCL morphologies may be associated with normal growth and development processes, such as fine porosity (Cole and Waldron 2019).

Early paleopathological studies of PCLs focused on the Southwest identified associations between lesion prevalence and age. In a study examining PH in Ancestral Pueblo skeletal samples, El-Najjar and colleagues (1976) suggested that PH rates were higher in children because of incompletely mineralized, thinner cranial vault bones, which provide limited space for marrow expansion. Lallo (1977:471) cited "synergistic" interactions between developmental physiology and iron deficiency as key factors in the development of PH, hypothesizing that individuals who experienced nutritional stress during the weaning period and those who experienced particularly rapid somatic growth (with its attendant energetic costs) would be more likely to develop lesions.

Although not focused on the Southwest, Stuart-Macadam (1985) used studies of bone marrow physiology to contextualize data on PCLs derived from clinical and anthropological sources. She suggested that greater bone plasticity in conjunction with limited capacity for marrow expansion increased the likelihood of lesion development in anemic juveniles. This study provided strong support for the theory that PCLs are representative of childhood episodes of stress—while calling into question the role of diet in their development.

Diet and Skeletal Alterations

The association of PCLs with nutrient deficiencies, such as iron, B_{12}, and folate, and resulting anemia (El-Najjar et al. 1975; Walker et al. 2009) may arise from a mischaracterization of a population's typical diet; for additional discussion, see Cadwallader and colleagues (2012). In the Southwest, the connection of PCLs to acquired anemia related to maize dependency likely derives from a series of work by El-Najjar and coworkers (1975, 1976). This research analyzed individuals from Basketmaker II–III and Pueblo I–V sites in Arizona and New Mexico, including those from Chaco Canyon (individuals from Chaco Canyon are included in our study).

The 1976 study examined 539 crania for evidence of PCLs and found that rates varied with ecological context: populations living in canyon bottoms exhibited higher rates of PCLs than those living on sage plains. The authors attributed this result to dietary variation, hypothesizing that maize-dependent populations with less access to faunal sources of iron were more likely to develop PCLs (El-Najjar et al. 1976). The subsequent observation that PCL frequencies increased with the introduction of maize agriculture in the Southwest (Berry 1984:264–265, citing Lallo et al. 1977) further established the relationship between maize consumption, iron-deficiency anemia, and PCLs in the archaeological literature.

Using El-Najjar and colleague's work as a reference, Ferguson (1980) concluded that iron-deficiency anemia was common in those who lived in Tijeras in New Mexico and that diarrheal disease or prolonged breastfeeding without iron supplementation were likely factors in its development. Merbs and Miller's (1985) collection of research papers includes four chapters devoted to PCLs, in which

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iron-deficient maize-based diets are consistently cited as contributors to lesion development. For example, Walker (1985) examined PCLs in samples from sites also represented in our study: Chaco Canyon and Tijeras. In addition to iron-deficient diets, Walker hypothesized that other factors, including breastfeeding, parasitism, and other nutritional deficiencies, may have contributed to the development of PCLs in Southwest archaeological contexts. These works were at the forefront of research at the time they were written and were designed to identify stressed individuals within populations, the underlying environmental factors that contributed to PCL formation, or both.

Although PCL frequencies are thought to increase with the introduction of maize agriculture (Berry 1984:264–265, citing Lallo et al. 1977), their presence and prevalence do not necessarily have a relationship with maize consumption or reliance (Reinhard 1988; Stodder 1989; Ubelaker 1992; Walker 1986; see also Rothschild 2012). In addition to the dietary implications of maize agriculture, other changes occur with its adoption, including population growth and population aggregation.

Maize was a dietary staple for many Southwest groups (Brand 1994; Hard et al. 1996; Holliday 1996; Mays 2008; Minnis 1989). Although there are many problems with a diet solely dependent on maize (if such a diet exists), the nutritional-deficiency argument centers on its iron content. Maize is low in iron and can limit iron bioavailability—the body's ability to absorb iron. A study by Moore (1968) estimates iron absorption from maize at 5%. The presence of phytic acid content in maize can also limit iron bioavailability by acting as a chelating agent. Iron bioavailability differs based on the type of iron in food sources. Heme iron from meat, fish, and other seafood is the most bioavailable. Nonheme iron is available in grains, seeds, nuts, and dark leafy greens.

Even when maize is a dietary staple, other food items are incorporated into the diet (Cadwallader et al. 2012), as was the case in the ancient Southwest (Adams 2008:Table 4.3; Huckell and Toll 2004: Tables 3.1–3.5). Southwest groups ground maize using limestone or treated it with lime or ash, enhancing its mineral and nutritional content (Beck 2001:190; Ezzo 1994:271; Huss-Ashmore et al. 1982:400; Snow 1990:293). The nutrient content of maize is also enhanced by foods consumed alongside it, including beans, squash, tubers, prickly pear, goosefoot, yucca, pine nuts, amaranth, and beeweed (Anschuetz 2006; Kellner and Schoeninger 2007; Kinder et al. 2017; Reinhard 1992; Reinhard and Danielson 2005; Watson 2008). The diet also included meats and animal proteins from turkeys, rabbits, prairie dogs, artiodactyls, and fish (Badenhorst et al. 2019; Dombrosky et al. 2020; McKusick 1982; Rawlings and Driver 2010; Snow 2002).

Methods and Case Study

We examined the distribution of PCLs by individual age and the percent frequency of PCLs alongside evidence for diet from multiple Southwest sites and locales. We expected that PCL types would show patterning by age and that evidence for micronutrient deficiency (scurvy or rickets) would be lacking from skeletal remains, as supported by the literature and by faunal, coprolite (including paleoethnobotanical), and other dietary data.

We included data from 86 Ancestral Pueblo individuals under age 16 who lived in what is now New Mexico between AD 1000 and 1400. We selected this age-restricted assemblage because PCLs develop in children, although adults may maintain evidence of PCLs throughout their lives (Brickley 2018; O'Donnell et al. 2022; Stuart-Macadam 1985), and PCLs in children are unlikely to have undergone extensive remodeling. It is worth noting, however, that remodeled and remodeling PCLs are found in children, including those Ancestral Pueblo individuals in our study (O'Donnell 2019). Lallo (1977) argued that skeletal remodeling is a methodological problem in any study of PCLs, and the rate and extent to which they remodel under varied environmental conditions remain uncertain. In addition to influencing assessments of PCL presence and prevalence, remodeling may affect assessments of the morphological characteristics of lesions.

The Museum of Indian Arts and Culture (MIAC) staff conducted consultation on O'Donnell's behalf. Permission was granted for all data included here; see O'Donnell, Valesca Meyer, and Ragsdale (2020) for a detailed explanation of the process. All methods used here are nondestructive. The case study is subject to archaeological issues, including small assemblage size, but power analysis indicates a sufficient (0.83; alpha = 0.05) sample for logistic regression estimates.

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Cultural Context

Most individuals included in the case study lived between AD 1100 and 1400, which saw significant demographic and environmental disturbances, large-scale migrations, regional depopulation, and aggregation (Cordell 1995; Kohler et al. 2010). In the late 1200s, the Four Corners region experienced depopulation, often attributed to the "Great Drought" (Schlanger and Wilshusen 1993), although this explanation is likely too simplistic (Crown et al. 1996; Kohler et al. 2010; Ortman 2010; Roney 1995; Schillaci and Lakatos 2016; Varien 2010). The population had survived severe droughts equivalent in intensity or worse than the Great Drought, and some areas were buffered from the drought's effects (Benson et al. 2007).

There is ethnographic, archaeological, and bioarchaeological evidence for inequity and unequal distribution of resources in Puebloan society (Eggan 1950; Levy 1992; Martin and Osterholtz 2016). None of the individuals in this sample with associated burial information had grave goods or burial treatment indicative of elevated social status (O'Donnell and Moes 2021). However, some individuals and groups likely experienced heightened stress related to social status, sex, or age (see Table 1 for assemblage information).

Skeletal Analysis and Data Collection

Skeletal analyses were done using standard methods (Buikstra and Ubelaker 1994; Ortner 2003; see O'Donnell 2019). Age was estimated using dental and skeletal development (AlQahtani et al. 2010; Baker et al. 2005; Buikstra and Ubelaker 1994; Scheuer and Black 2000, 2004). Sex estimates are not possible in prepubertal children without doing destructive analyses (O'Donnell et al. 2017; Scheuer and Black 2000), so no attempts at sex estimation were made.

Each individual included had at least one observable eye orbit; PCL morphology scoring followed Stuart-Macadam (1991a:Figures 9.3a, 9.3b). Table 2 and Figure 1 provide descriptions and depictions of morphology, respectively.

Expansion of the diploic space, identified in dry bone when the marrow spaces are enlarged, was recorded. Expansion was observable in the orbit roof of six of 32 individuals and in the vault bones of 10 of 62 individuals; for additional depictions, see Figure 2, Grauer (2019:Figure 14.24), and Brickley and coauthors (2020:Figure 9-2). Because expansion was recorded in bones broken postdeposition, there may be some comparability issues. However, marrow hyperplasia (thickening of dry bone) is observable at postdepositional breaks (Mays 2012:291), and expansion outside the norm is identifiable (see O'Donnell et al. 2023:Figure 1B–1D).

Assessing Diet in the Southwest

In addition to the literature review, we aggregated information from paleofeces (coprolite)/paleoethnobotanical studies to characterize the Ancestral Pueblo diet (see Table 3). The dates for coprolites range from 2900 BC to AD 1450, with the majority of coprolites being from PII (AD 900–1150) and PIII (AD 1150–1325). Not all dietary components are equally represented in coprolites. Some food items, such as beans, squash, and meat, are wholly digested or otherwise difficult to identify in coprolites (Stiger 1977:16). In contrast, maize is not completely digested and may be overrepresented in coprolites (Reinhard 1988:144, 147). However, coprolites enable the direct assessment of human diet and parasitism via the presence of macroscopic (e.g., animal bones, seeds) and microscopic inclusions (e.g., pollen, phytoliths; Reinhard and Bryant 1992). Small animal bone fragments in coprolites provide evidence for meat consumption (Clary 1983; Gillespie 1981; Reinhard and Bryant 1992). The accuracy of the frequencies of coprolites is dependent on the number of observations (presented in Supplemental Table 2).

Data from multiple studies (Table 3; Supplemental Tables 2 and 3) were used to plot temporal patterns of PCL presence/absence. Analyses of lesion morphology were conducted on the 86 individuals in the case study.

Analytical Methods

Logistic regression in Stata 18 (StataCorp 2017) was used to estimate odds ratios (OR) for the associations between PCLs and estimated age at death. OR are commonly used in clinical research to

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Table 1. Individuals Included in This Study; Counts of Individuals by Site and Age Group (One-Year Intervals).

Region	Dates	Midpoint	Number of Individuals	Age Range and Number of Individuals
East Mesa Verde [also called Totah]	1000–1300	1150	11	2–2.9 years: 4 5–5.9 years: 2 7–7.9 years: 1 9–9.9 years: 2 10–10.9 years: 1 11–11.9 years: 1
San Juan Basin	800–1325	1063	10	6 months–11.9 months: 2 1–1.9 years: 1 2–2.9 years: 1 3–3.9 years: 3 4–4.9 years: 1 5–5.9 years: 1 9–9.9 years: 1
Northern Rio Grande	1050–1600	1325	13	6 months–11.9 months: 1 1–1.9 years: 1 2–2.9 years: 1 3–3.9 years: 1 4–4.9 years: 2 5–5.9 years: 3 6–6.9 years: 1 8–8.9 years: 3
Gallina District	900–1300	1100	7	6 months–11.9 months: 1 1–1.9 years: 1 4–4.9 years: 1 5–5.9 years: 1 8–8.9 years: 1 11–11.9 years: 1 13–13.9 years: 1
Middle Rio Grande	1262–1600	1431	38	6 months–11.9 months: 11 1–1.9 years: 5 2–2.9 years: 8 3–3.9 years: 6 4–4.9 years: 2 7–7.9 years: 2 8–8.9 years: 2 13–13.9 years: 2
Mogollon (Jornada)	700–1450	1075	4	6 months–11.9 months: 2 4–4.9 years: 1 6–6.9 years: 1
Mogollon (Mimbres)	1000–1450	1225	2	7–7.9 years: 1 9–9.9 years: 1
Rio Abajo	1250–1450	1350	1	6 months–11.9 months: 1
Total Individuals			86	6 months–11.9 months: 18 1–1.9 years: 8 2–2.9 years: 14 3–3.9 years: 10 4–4.9 years: 7 5–5.9 years: 7 6–6.9 years: 2 7–7.9 years: 4 8–8.9 years: 6 9–9.9 years: 4 10–10.9 years: 1 11–11.9 years: 2 13–13.9 years: 3

Note: All individuals included in this study are from pre-Spanish contact site components.

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Table 2. Lesion Types (Morphology) and Definitions Used in the Study.

Score	Descriptions
0	Normal bone (no lesion)
1	Capillary-like impressions
2	Scattered, small foramina (1 mm or less)
3	Large and small scattered foramina (1 mm or greater)
4	Foramina linked in trabecular structure (coral-like)
5	Outgrowth in trabecular form from the outer table (coral-like)
6	New bone formation on outer cortex
Cranial Vault and Orbit – Thickening, Thinning, Marrow Hyperplasia	
Normal	Normal diploë (marrow spaces) and thickness
Marrow Hyperplasia	Characterized by enlargement of spaces in the diploë (expansion of marrow space), which would have been caused by marrow expansion. The enlarged spaces would have been occupied by bone marrow in life (see also Brickley et al. 2020:Figure 3–6c).
Lamination	Characterized by multiple, thin layers of bone that is "compact" between the diploë; can be accompanied by expansion of the bone or not (vault only).
Thickening	Thickening of the orbit roof or vault bones out of character with the age of the individual, which could be due to hyperplasia, new bone, or another cause.
Thinning	Can refer to cortical thinning, caused by marrow hyperplasia or thinning of the diploë (see also Brickley et al. 2020:Figure 9-2b). No thinning was observed in broken cross sections in this study.

Note: See Figures 1 and 2 for illustrations of lesion types and bone cross sections. These scores were also used for porotic hyperostosis (see Stuart-Macadam 1991a:Figure 9.3a, 9.3b; orbits schematics follow Figure 9.3b).

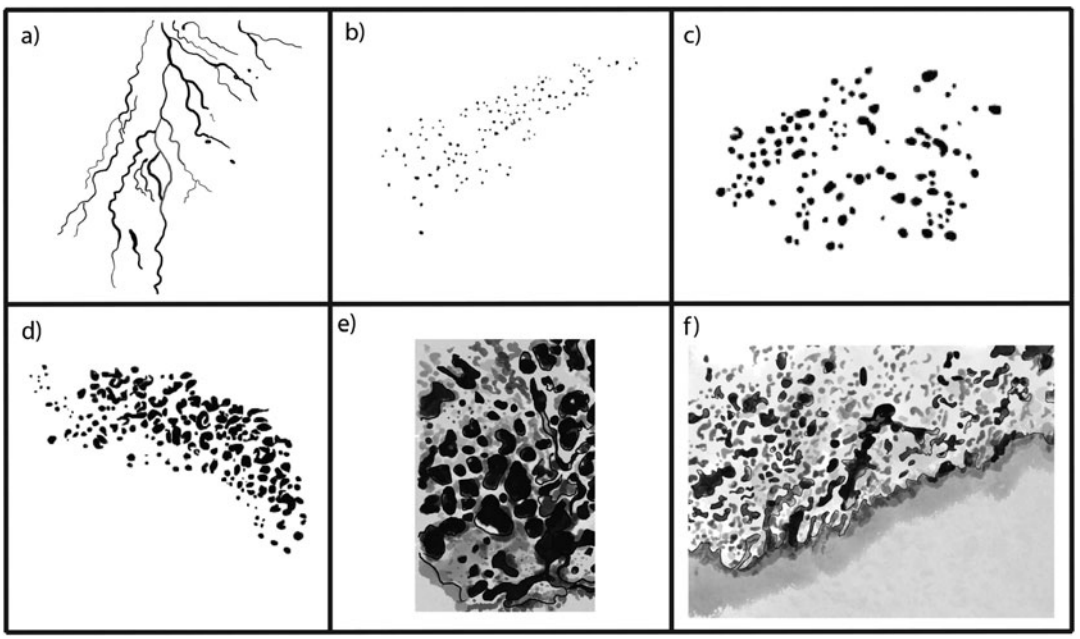

Figure 1a. Illustrations of CO and PH morphology (Types 1–6) described in Table 2. 1a-a Type 1, capillary-like impressions; 1a-b Type 2, scattered, small foramina; 1a-c Type 3, large and small scattered foramina; 1a-d Type 4, foramina linked in a trabecular structure; 1a-e Type 5, trabecular outgrowth from the outer table; 1a-f Type 6, new bone deposited on outer cortex. All illustrations by the first author.

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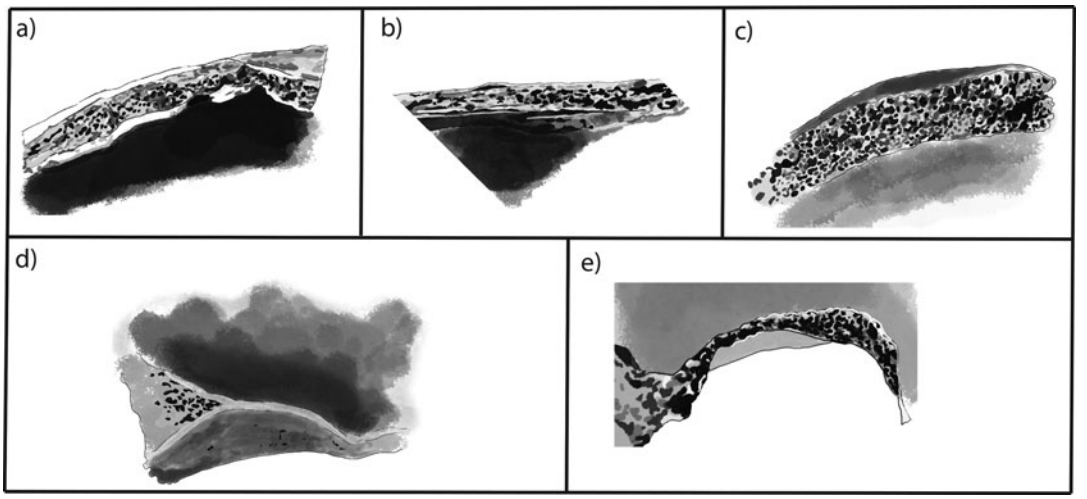

Figure 1b. Illustrations of cross sections of vault (Figure 1b-a and 1b-c) and orbit bones (1b-d, 1b-e). (1b-a) Normal vault cross section; (1b-b) lamination of diploë without expansion of the marrow space; (2c) expansion of the marrow space and thickening, coupled with thinned outer/inner cortices; (1b-d) normal orbit; (1b-e) orbit with expansion of the marrow space accompanied by thickening of the bone. Orbit illustrations are from the posterior (looking into the endocranium). All illustrations by the first author. (See also Brickley and colleagues 2020:Figure 9-2; O'Donnell et al. 2023:Figure 1).

examine the odds of developing an outcome following an exposure (e.g., lung cancer in those who smoke). Here, we use OR to examine the odds of having a lesion characterized by certain morphology, such as Type 6 (outcome), based on individual age at death (exposure). Logistic regression was done with regions pooled by age at death, rather than focusing on individual regions. For region- and site-level analyses, see O'Donnell (2019:207–214).

An OR > 1 indicates greater odds of having a lesion, an OR < 1 indicates lower odds of having a lesion, and an OR = 1 indicates no difference in odds of having a lesion. OR are presented in forest plots (coefplot; Jann 2013), which provide visual representations of the analysis results. A red line is placed at 1. Significant results to the right of the line indicate increased odds of having an outcome, whereas those to the left of the line indicate decreased odds of having an outcome.

The regression formula is

$$\Pr\left(Lesion\ Type_i = 1\right) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 age_i + u_i)}}$$

Index i is an individual observation, β is the coefficient estimated using regression, *age* is individual age at death, and u is the error term. *Lesion Type* is the outcome of interest (*Lesion Type* = 1 if the outcome is observed; Lesion Type= 0 if the outcome is not observed). For example, Lesion Type 5 can be "0" absent or "1" present. The variable age^2 was included in the logistic regression analyses to account for nonlinear relationships between age at death and PCL presence/absence but was insignificant for all analyses, so results are provided for age alone.

Balloon plots showing the frequency of dietary inclusions and PCLs (size and color of the "balloon") were made using RStudio's ggplot (RStudio Team 2016). The ggplot package was used to plot density and smoothed percent frequency curves.

Results

Diet in the Southwest

Maize was ubiquitous throughout time, but there was substantial dietary variety (Figure 2a). Squash and beans are found at lower frequencies than maize, likely due to complete digestion. Chenopods and amaranth (cheno-ams) are in several categories in Figure 2a (cheno-ams, goosefoot). Other

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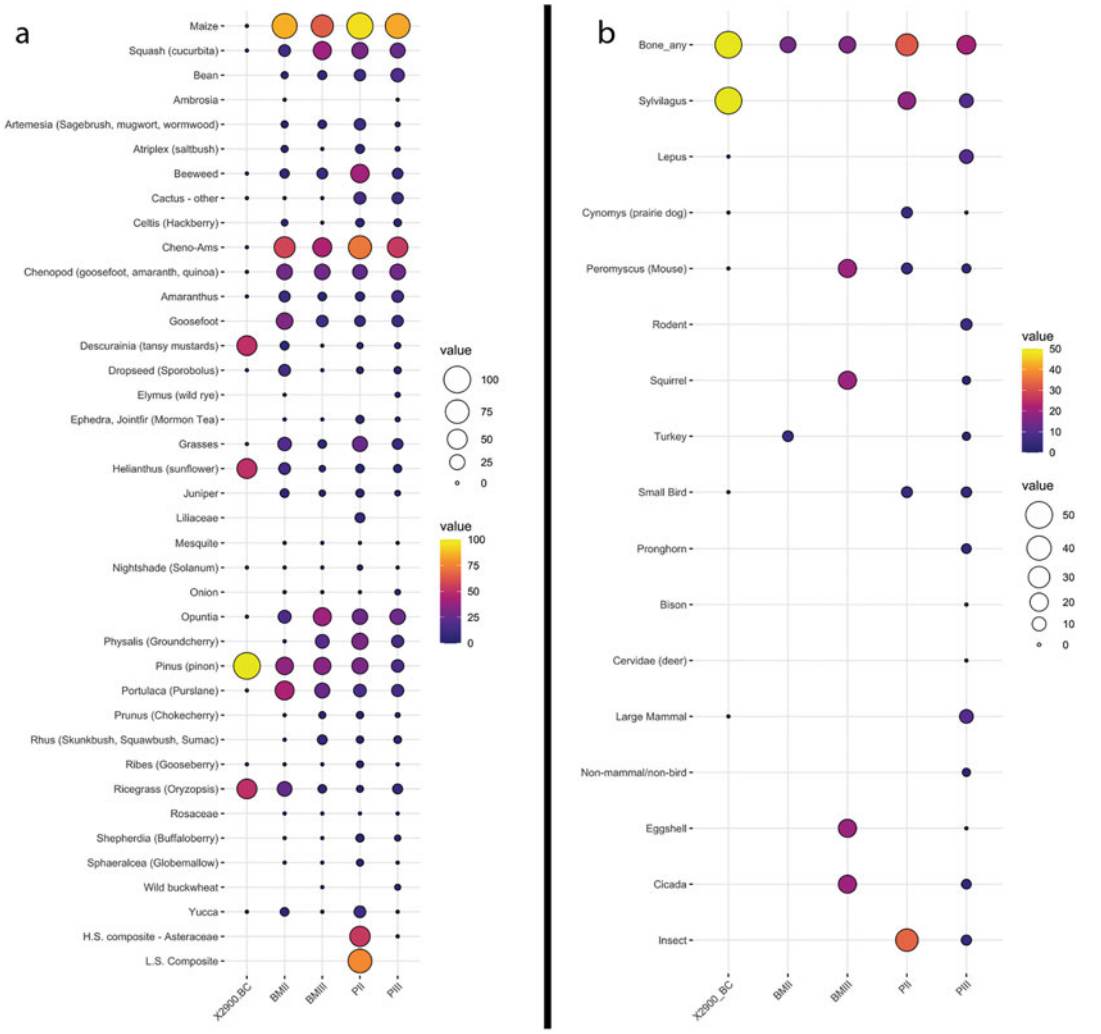

Figures 2a–2b. Balloon plots show the percent frequency of coprolites with pollen or plant (2a) / animal remains (2b) across time. The "balloon" increases in size with increasing frequency, and the color increases in temperature following that same pattern. For the number of coprolites per site, see also Supplemental Figure 1 and Supplemental Table 2; references for data are shown in Table 3. Abbreviations: BMII (1500 BC–AD 50), BMIII (AD 500–750), PI (AD 750–900), PII (AD 900–1150), PIII (AD 1150–1325), PIV (AD 1325–1550). (Color online)

common food items are pine nuts (*Pinus*), grasses, beeweed (*Cleome*), sunflowers (*Helianthus*), purslane (*Portulaca*), and prickly pear (*Opuntia*). Figure 2b shows that large and small game animals, including rabbits, were part of the diet across time.

PCL Presence and Morphology, Age at Death, and Diet

The most common PCL morphology is Type 2. Type 4 morphology in the orbit and on the parietal bones shows a bimodal distribution (Figures 3a, 3c). The frontal bone is the least likely to exhibit lesions (Figure 3b). Figures 3 and 4 demonstrate that the probability of having PCLs decreases with age: their presence is associated with younger ages at death in this sample (Figure 4a). CO characterized by "simple porosity" (Type 2) is associated with younger ages at death (Figure 4b, purple circle). PH characterized by Type 6 on any vault bone is associated with younger ages at death (Figure 4b, open blue circle). In analyses examining the relationship between PCL morphology and age in individuals under 10 years old (Figure 4c), we find similar results. There is one notable difference: in

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Table 3. Sources of Coprolite Dietary Data.

Site or Region	State	Dates*	Data Sources
Coprolite Studies			
Antelope House	AZ	PII (1000) PII/PIII (1090–1300) PIII (1150–1300)	Fry and Hall 1986; Stiger 1977; Reinhard 1988: Table 5; Sutton and Reinhard 1995; Williams-Dean and Bryant 1975
Inscription House	AZ	PIII (1150–1300)	Brand 1994; Reinhard 1988:Tables 9 and 22; Stiger 1977
Ventana Cave	AZ	PII/PIII (1000–1450)	Reinhard and Hevly 1991
Mesa Verde	CO	BMIII (1–750) PIII?	Stiger 1979
Step House	CO	BMIII (500–750) PIII (1150–1300)	Brand 1994; Reinhard 1988:Table 5; Stiger 1977
Hoy House	CO	PII (1090–1150) PIII (1250)	Reinhard 1988:Table 5; Scott 1979; Stiger 1977
Lion House	CO	PIII (1240)	Stiger 1977:26, Table III
Atlatl Cave	NM	2900 BC	Clary 1983
Bat Cave	NM	BMII-PII (200–1000)	Trigg et al. 2000
Pueblo Alto	NM	PII (1020–1100)	Clary 1983, 1987
Pueblo Bonito	NM	PII (850s–1100s)	Clary 1983
Kin Kletso	NM	PIII (1125–1300)	Clary 1983
Chaco Canyon	NM		Clary 1983
Aztec Ruins – West Ruin	NM	PII (1090–1105)	Cummings et al. 2009; Stiger 1977
Salmon Ruin	NM	PII (1090–1150)	Reinhard 1988:Table 5, 2006
Turkey Pen Ruin	UT	BMII (1–400)	Battillo 2017, 2019
CO and PH Studies			
Antelope House	AZ	BMII–BMIII (400–700); PII/PIII (900–1300)	El-Najjar 1986
Canyon de Chelly	AZ	BM (300–700) PII/PIII (700–1300)	El-Najjar et al. 1976
Canyon del Muerto	AZ	Early BM (700–1500 BC)	Zaino 1967

(*Continued*)

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Table 3. Sources of Coprolite Dietary Data. (*Continued.*)

Site or Region	State	Dates*	Data Sources
Carter Ranch	AZ	PIII (1100–1225)	Danforth et al. 1994
Grasshopper Pueblo	AZ	PIII/PIV (1225–1450)	East 2008:Table 100
Houck	AZ	PI/PII (900–1350)	Zaino 1967
Inscription House	AZ	PIII (1250–1300)	El-Najjar et al. 1976
Kayenta	AZ	PII; PIII	Stodder 1989:Table 42
Navajo Reservoir	AZ	PI–PIII (700–1100)	El-Najjar et al. 1976
Oak Creek Pueblo	AZ	PII– PIV (1125–1400)	Taylor 1985
Point of Pines	AZ	PII–PIV (1240–1450)	East 2008:Table 100
Turkey Creek Pueblo	AZ	PIII (1225–1286)	East 2008:Table 100
Animas La–Plata	CO	PI (750–900)	Perry et al. 2010
Black Mesa	CO	BM–PII (700–1100)	Stodder and Martin 1992
Mesa Verde	CO	PI–PIII	Stodder 1989, 1996; Stodder and Martin 1992:Tables 2 and 3
Sand Canyon Pueblo	CO	PIII (1240s–1280s)	Kuckelman and Martin 2007
Chaco Canyon	NM	PII (700–1100)	El-Najjar et al. 1976
Pueblo Bonito	NM	PII (850–1100)	Harrod 2012; Marden 2011
Chaco Small Sites	NM	PII/PIII (900–1178)	O'Donnell 2019
Kin Kletso	NM	PIII (1125–1300)	O'Donnell 2019
Pueblo Alto	NM	PII (1020–1100)	Akins 1987
Kin Bineola	NM	PII (940–1120)	Harrod 2012
Salmon Ruin	NM	PII (900–1100); PII (1090–1150)	Doebley and Bohrer 1983; Shipman 2006; Stodder and Martin 1992:Table 5
Aztec Ruin	NM	PII (1090–1105) (West Ruin)	Harrod 2012
Arroyo Hondo	NM	PIII–PV (1300–1600)	Palkovich 1980, 1985

(*Continued*)

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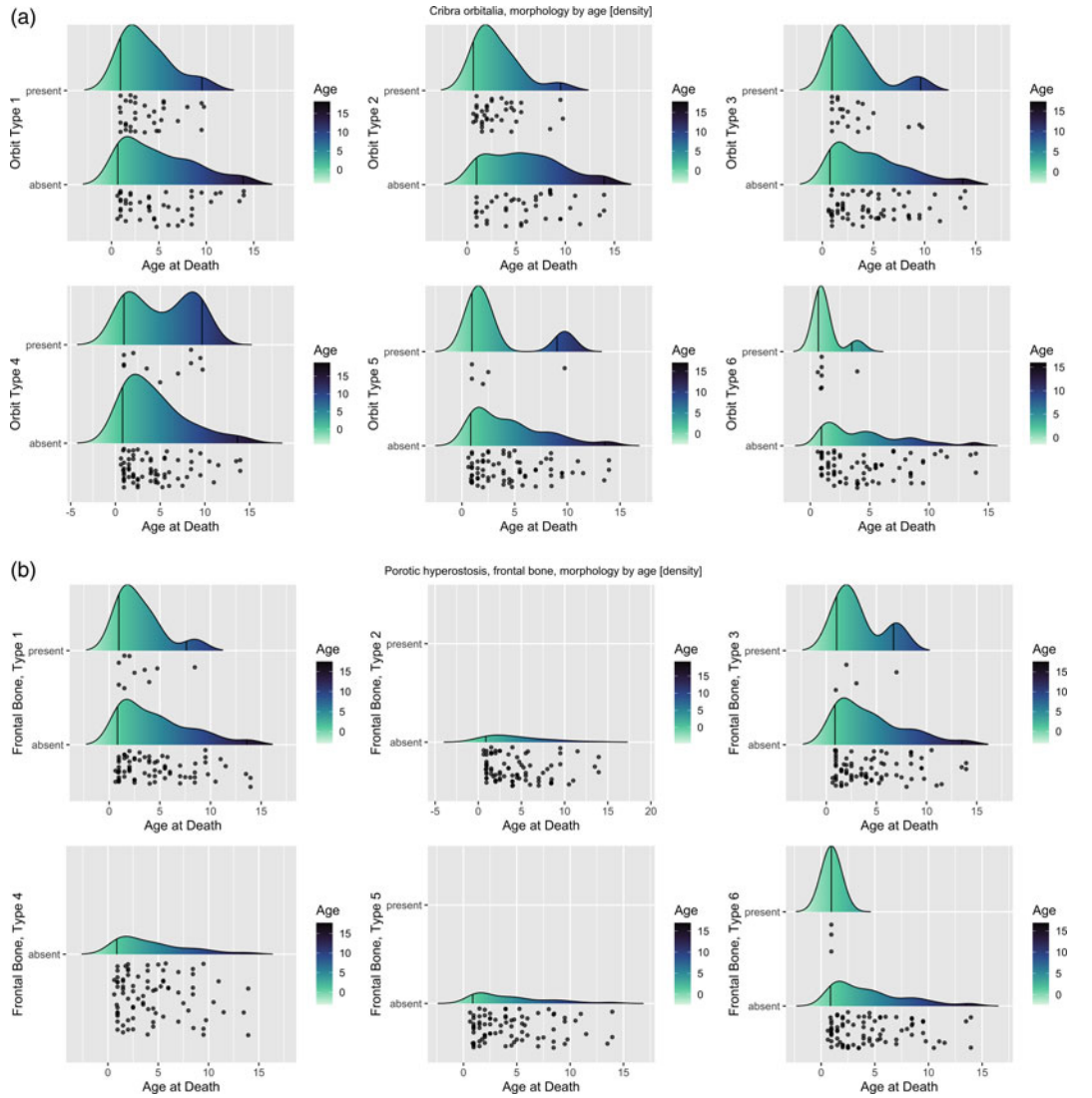
Table 3. Sources of Coprolite Dietary Data. (*Continued.*)

Site or Region	State	Dates*	Data Sources
Casamero	NM	PII (900–1150)	O'Donnell 2019
Gran Quivira	NM	PIII–PV (1300–1600)	El-Najjar et al. 1975; Stodder and Martin 1992
Hawikku	NM	PIII–PV (1300–1600)	Stodder 1996:Table 5.2
La Plata Highway	NM	PII/PIII (1000–1300)	O'Donnell 2019
Pa'ako	NM	PII–PV (1100–1600)	Ferguson 1980
San Cristobal	NM	PIII–PV (1300–1600)	Stodder 1996:Table 5.2
Northern Rio Grande	NM	PII–PV (1050–Historic)	O'Donnell 2019
Middle Rio Grande	NM	PIII/PIV (1260–1400)	O'Donnell 2019
Gallina District	NM	PII/PIII (900–1300)	O'Donnell 2019
Jornada Mogollon	NM	BM–PIV (700–1375)	O'Donnell 2019
Mimbres Mogollon	NM	PII/PIII (1000–1325)	O'Donnell 2019
Time Period	*Midpoint*	*Date Range*	
Basketmaker (BM) II	775 BC	1500 BC–AD 50	
BMIII	AD 625	AD 500–750	
PI	AD 825	AD 750–900	
PII	AD 1025	AD 900–1150	
PIII	AD 1238	AD 1150–1325	
PIV	AD 1438	AD 1325–1550	

Notes: These data are used in Figure 2; pollen studies generally required >=200 grains; CO and PH data are used in Figure 6. The bottom portion of this table provides the date ranges used along with midpoints. Refer to Supplemental Tables 2 and 3 for counts.

* Although there are some difficulties with using the Pecos Classification to compare areas throughout the Southwest (not all sites or groups fit neatly into the categories), it is used here to generalize trends across time and space.



Figures 3a–3b. These figures provide density curves by age at death for lesion Types 1–6. (3a) CO, orbit; (3b) PH, frontal bone; (3c) PH, parietal bones; (3d) PH, occipital bone. Along the x-axis at the bottom of each figure are points indicating each individual in the assemblage. (Color online)

the younger age group, Type 3 PH on the parietal bone is associated with younger ages at death (Figure 4c, orange triangle).

CO follows no discernible pattern across time, and PH increases as time progresses (Figure 5). However, the CO or PH category in this figure represents instances where authors pooled the lesions (implying an inferred shared etiology). If CO and PH had been broken into two categories, the frequency patterns observed might have been altered.

Few individuals in the case study had a suite of skeletal changes consistent with scurvy ($n = 3$; 3.5%) or rickets (no identified cases), and neither produced pathognomic skeletal alterations (Brickley et al. 2020:53, 95).

Discussion

PCL morphology should be reflective of lesion etiology (e.g., Brickley 2024; O'Donnell, Hill, et al. 2020). We found that expansion of the diploic space (which would be reflective of anemia) is rare,

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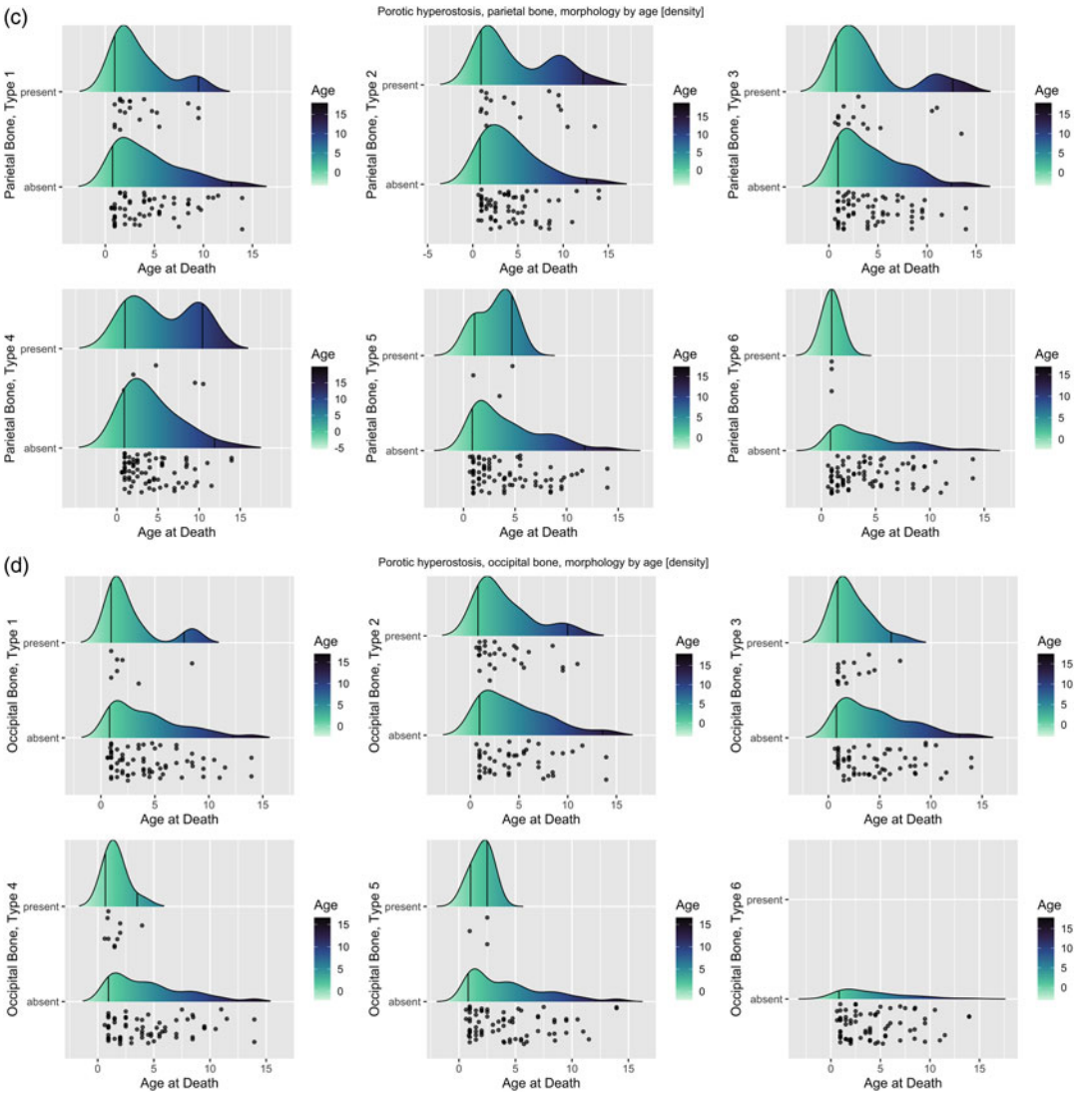

Figures 3c–3d. These figures provide density curves by age at death for lesion Types 1–6. (3c) PH, parietal bones; (3d) PH, occipital bone. Along the x-axis at the bottom of each figure are points indicating each individual in the assemblage. (Color online)

whereas lesions characterized by fine porosity are more common. The diversity of PCL morphology implicates varied processes in their formation. These could include immune responses such as inflammation, infections, neoplasia, nutrient deficiencies, and trauma, although no skeletal injuries were identified in our study. Considering morphology in concert with archaeological evidence for diet can help researchers identify and rule out potential diagnoses and underlying biological mechanisms that promote lesion formation (Brickley et al. 2020:249; Mays 2018a).

Early studies of PH in the Southwest, which largely attributed lesion formation to maize-dependent diets, noted that ecological factors and dietary variation likely influenced lesion rates (see El-Najjar et al. 1976; Walker 1985). We highlight these studies because the present sample overlaps with theirs. Walker (1985) and El-Najjar and colleagues (1976) considered the potential role played by other conditions—parasitism and, for adults, breastfeeding—in the development of anemia, but limited available evidence led El-Najjar and coauthors to conclude that dietary factors were a more likely cause of lesion formation in the Southwest. More recent ethnobotanical and coprolite studies provide a more nuanced

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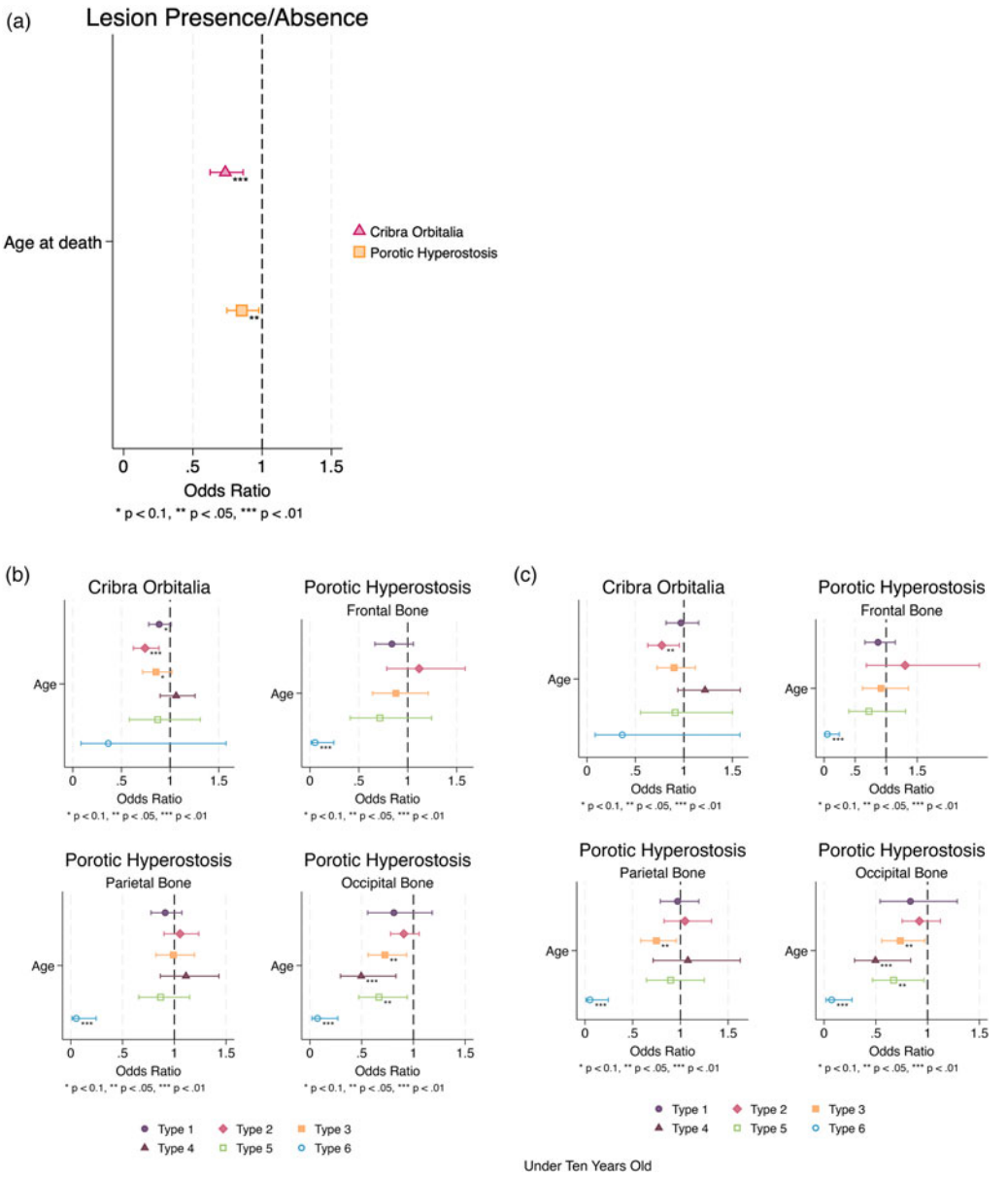

Figures 4a–4c. Each figure presents odds ratios depicting the relationship between PCL location, type, and age at death in individuals 6 months to 15 years; Figure 4a presents results for CO (triangle) and PH (square) presence, regardless of morphology, by age at death. Figures 4b and 4c present the same analyses, but Figure 4c provides results for individuals under ten years of age. These analyses focus on lesion types and age at death. Type 1 is represented with a filled circle (purple), Type 2 with a red diamond, Type 3 with an orange square, Type 4 with a purple triangle, Type 5 with an open green square, and Type 6 with an open blue circle. Significance values are provided for "p" at three levels: 0.01, 0.05 (threshold for statistical significance) and 0.1. $p \leq 0.1$ is provided for the reader as some of these values are $p < 0.07$ but $p > 0.05$. Lines represent 95% confidence intervals. Of note, no individuals with type 4 PH were observed for the frontal bone. (Color Online)

picture of Southwest dietary variation and risks posed by soil and water-borne parasites, contextualizing observed PCL rates and morphological variation.

Although this article focuses on the US Southwest, its findings are of relevance to those who study human health and stress in other temporal and geographic contexts. For example, other studies such as that of Cadwallader and colleagues (2012) indicated that the results of paleodietary reconstructions should be interpreted with care. The presence and meaning of PCLs should be interpreted similarly.

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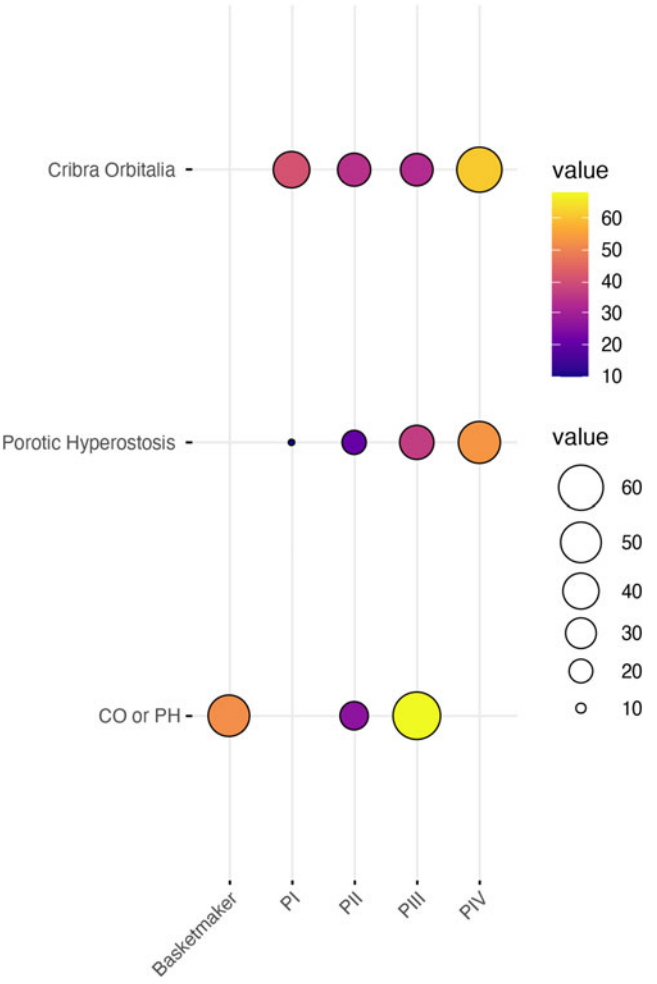

Figure 5. Balloon plot shows the percent frequency of CO and PH across time. The "balloon" increases in size with increasing frequency, and the color increases in temperature following the same pattern. References for data are shown in Table 3. Abbreviations: BMII (1500 BC–AD 50), BMIII (AD 500–750), PI (AD 750–900), PII (AD 900–1150), PIII (AD 1150–1325), and PIV (AD 1325–1550). (Color online)

PCLs are found around the globe in varied archaeological settings (Carlson et al. 1974; Jatautis et al. 2011; Keenleyside and Panayotova 2006; Obertová and Thurzo 2004; Schats 2023; Smith-Guzman 2015; Zaino and Zaino 1975). They have also been identified in contemporary populations (Anderson 2022; Beatrice and Soler 2016; Beatrice et al. 2021; O'Donnell, Hill, et al. 2020) and are emerging as markers of generalized stress in public health contexts (O'Donnell et al. 2024). If their presence is assumed to derive from a single cause, then we may overestimate the frequency of conditions historically implicated in PCL development—for example, iron-deficient diets—while failing to recognize the multifactorial nature of PCL development and the role of other potential causative factors: dietary, immunological, and social. PCLs may be interpreted as indicators of stress suffered by a developing individual, but unless their morphology is used to diagnose the cause, they should not be seen as hallmarks of specific conditions.

Reconstructed Southwest Diet

Coprolite, paleoethnobotanical, and faunal studies demonstrate a varied and, in general, nutritionally adequate diet in the Southwest, with a variety of cultivated and wild foods supplementing maize (Durand and Durand 2008; Huckell and Toll 2004; Woosley 1980). Maize on its own is nutritionally incomplete, and the consumption of wild food sources provides critical micronutrients. For example, rabbits, an essential and ubiquitous dietary component (Brand 1994:39; Coltrain and Janetski 2013; Durand and Durand 2008:103; Holliday 1996), provide vitamin B_{12} and other nutrients, including

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heme iron (Nistor et al. 2013; USDA 2021). Fish, which were also eaten, provide heme iron and also increase the nonheme absorption of iron from maize (Hutchinson et al. 2007; Layrisse et al. 1968).

Many wild plants are higher in iron, protein, and other micronutrients than maize. Pine nuts are a seasonally available source of protein, folate, and lysine. Yucca, juniper berries, opuntia, and wild mustard are also sources of vitamin C (Barriada-Bernal et al. 2018; Truesdale 1993). Cheno-ams were noted at high frequencies in our survey, rivaling those of maize (Figure 2a). Amaranth is high in protein and contains higher levels of calcium, iron (29% of the recommended daily intake in one cup), magnesium, and sodium than other cereal grains (Ajmera 2018; Stallknecht and Schulz-Schaeffer 1993). It is also a good source of carotenoids (Duya et al. 2018), which increase iron uptake and ferritin synthesis (García-Casal 2006). The fungus *Ustilago maydis* (*huitlacoche*, or corn smut), which has higher protein content than maize, may have also been consumed (Battillo 2018).

Infants and children are more likely to develop PCLs than adults, but their diets are more challenging to assess in Southwest archaeological contexts. Coprolite studies do not specify whether adults or children deposited them. Isotopic studies involving infants and children are limited in number and sample size and largely restricted to the Basketmaker II and III periods. These suggest that the timing of weaning was highly variable and that the diets of nursing infants were likely supplemented with weaning food such as maize gruel (Coltrain and Janetski 2013).

Ethnographic research provides further clues to Puebloan infant and child diets. Cahuilla (Southern California) babies were fed piñon nuts ground up and mixed with water (Moerman 2010:184). The Haudenosaunee (Iroquois) did something similar with butternut (Moerman 2010:131), and Hopi (Pueblo) children were given small bits of food, including corn, mutton, and fruit (Eggan 1943:363). These studies suggest that supplemental foods provided to infants were varied and likely extended beyond maize gruel. In some cases, Pueblo babies were nursed for up to three years (Gonzalez 1974). In Ancestral Pueblo archaeological contexts, prolonged weaning, diarrheal diseases, and resultant iron deficiency have been implicated in the elevated PCL rates observed in infants (Kunitz and Euler 1972). However, maternal buffering via nursing promotes the delivery of key nutrients and immune factors during episodes of nutritional stress and may limit the development of dietary deficiencies and diarrheal disease—if mothers' micronutrient stores are not substantially depleted (Thayer et al. 2020).

It is impossible to know whether the ethnographic literature accurately reflects infant and child diets. However, we might infer that children in the Southwest were fed an adequate diet that included food items consumed by adults. This varied diet and the impact of maternal buffering render it unlikely that diets deficient in iron or vitamins (B_{12}, C, D, and others) are causes of all or even many of the lesions recorded and reported in the Southwest.

Of course, there were times of dietary, environmental, or social stress, which may have led to increased biological stress and potentially to PCL formation. During the mid- to late 1200s and early 1300s, there was significant demographic upheaval in northern New Mexico and the Mesa Verde region that caused environmental and social stresses, including migrations (Crown et al. 1996; Kohler et al. 2010). Psychosocial stress may contribute to PCL development (Beatrice et al. 2021), but studies of its impacts on PCL formation need to be conducted in children to address this hypothesis fully. Social and environmental stress might lead to dietary deficiencies, because maize production is reduced in periods of low rainfall or during years with reduced growing seasons.

Although some evidence of scurvy in the Southwest is provided by this study and others (Crandall 2014; Ortner et al. 2001), definitive skeletal evidence for nutrient deficiency is rare in the Southwest across time (East 2008; Ortner et al. 2001; Stodder et al. 2010:96) and in our analysis.

Bone Changes Seen in Anemia

Because anemias cause inconsistencies in hematopoiesis and hypoxia (WHO 2019a), they can result in alterations of bone. Hypoxia is associated with increased osteoclastic activity, suppressed osteoblastic activity, and bone loss (Hannah et al. 2021), and congenital anemias are associated with marrow hyperplasia. However, clinically identified bone changes in anemias and related diseases are rare (Aksoy et al. 1966; Lanzkowsky 1968; Sebes and Diggs 1979). Skeletal alterations, including PCLs,

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are not typically considered by clinicians when diagnosing anemia (Brickley and Morgan 2023:343). Furthermore, the severity of anemia is not a factor in lesion formation (Grauer 2019:517; Lanzkowsky 1968:25; Stuart-Macadam 1992:44). Not everyone who is anemic has skeletal involvement, and not everyone who has PCLs has anemia (Diggs et al. 1937; Lanzkowsky 1968; Wapler et al. 2004).

Wapler and coworkers (2004) found no histological indications of anemia in at least 56.5% (n = 333) of CO cases. Congenital anemias are more likely to induce marked skeletal alterations than acquired anemias (Lewis 2012; Brickley and Morgan 2023) but still do not produce alterations in all those afflicted (see also Brahmbhatt et al. 2017). PCLs are rarely observed in iron-deficiency anemia, with an incidence of 0.68; postcranial alterations occur more frequently (Agarwal et al. 1970).

A clinical connection between AoI and PCLs is uncertain, which has led to some disagreement in the bioarchaeological literature. Stuart-Macadam (1991b, 1992) is a proponent of the idea that infection, via AoI, caused many PCLs observed in bioarchaeological settings; see also Kent (2000). Other researchers found that AoI restricts erythropoiesis, so it should lead to bone loss, not marrow hyperplasia (Oxenham and Cavill 2010).

In our study, expansion was noted in 18.8% of individuals with postdepositional breakage of the orbit roof and 16.1% of individuals with postdepositional breakage of the vault bones. In a contemporary New Mexico mortality sample, 20.8% (133/475) had expansive lesions with pitting/porosity (orbit), but only 1.38% (5/362) were diagnosed with anemia, and none had iron-deficiency anemia (O'Donnell et al. 2023: Table 3). If expansive lesions are taken as evidence of iron-deficiency anemia, which would be ill-advised, then a small number of individuals included here may have suffered from iron deficiency.

The rarity of PCLs in clinical patients with iron-deficiency anemia (Agarwal et al. 1970), coupled with an adequate diet, renders it highly unlikely that it is responsible for most cases of PCLs observed in the Southwest. This does not discount a relationship between PCLs and nutrient deficiency/acquired anemias in all cases, but anemias should not be diagnosed solely through the presence of PCLs.

If Not Diet (in All Cases), What Explains PCLs?

Ancestral Puebloans and other Southwest groups likely experienced various health insults. Kent (1986:607) found that "maize, and diet in general, are at most only incidental causes of PH in the Southwest region." Instead, Kent (1986, 2000) argued that exposure to parasites, streptococcal infections, or other viral and bacterial agents could have caused PCLs, claims echoed by Stuart-Macadam (1992). Individuals sick for prolonged periods (a month or longer) have heightened odds of exhibiting PCLs (O'Donnell et al. 2023).

Respiratory Infection

Before 1900, pneumonia and gastrointestinal disease were common causes of child death (Mulholland 2007). Pneumonia can be sustained for a prolonged period (Haines et al. 2013), allowing time for skeletal lesions to form (Lindaman 2001; O'Donnell, Hill, et al. 2020). Kunitz (1970) suggested that respiratory infections were important contributors to patterns of morbidity and mortality observed in Ancestral Pueblo populations. Some environmental conditions that existed in the past increase the risk of pneumonia and respiratory illness. These include indoor air pollution caused by wood and dung burning known to occur in the Southwest (Jorgensen 1975; Windes and Ford 1996), crowded living conditions, parental smoking (WHO 2019b), and exposure to dust and pollen (Lambert 2002).

There is growing support for an association between PCLs and respiratory illness, although mechanisms underlying this relationship remain unclear. O'Donnell, Hill, and coworkers (2020) associate PCLs with respiratory illness in a contemporary mortality sample but stress that not every PCL is evidence of respiratory infection. Other studies found similar results; Gomes and colleagues (2022) and Anderson (2022) found associations between symptomatic tuberculosis and CO in a contemporary Bolivian population. Respiratory infections, including tuberculosis, bronchitis, and pneumonia, likely occurred in the Southwest. Although rare, tuberculosis was reported in several Southwest locales (East 2008; Lambert 2002; Ortner 2003; Stodder and Martin 1992). The reported cases likely represent a minimum number of infections (Roberts and Buikstra 2019).

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When taken in conjunction with dietary data, some PCL morphology may support assertions that infection and associated inflammation (perhaps AoI) are responsible for PCL development (Kent 1986, 2000; Stuart-Macadam 1991b). Although AoI is likely not responsible for expansive lesions (Oxenham and Cavill 2010), it could cause porosity, because inflammatory processes are linked to bone loss (Epsley et al. 2020).

Parasitism and Gastrointestinal Disease

There are no diagnostic criteria for identifying parasitism or gastrointestinal disorders in a skeleton. However, there is direct evidence for intestinal parasites in the Southwest from coprolite studies (Fry and Hall 1986; Reinhard 2008a, 2008b; Siqueira et al. 2010). Pinworm (*Enterobius vermicularis*) and human whipworm (*Trichuris trichiura*) eggs have been identified in coprolites recovered from Ancestral Pueblo sites in Chaco Canyon (Paseka et al. 2018). Parasitic infection was likely common among Ancestral Puebloans (Reinhard 2008a, 2008b) and may have been a consequence of cultural development related to agriculture (Paseka et al. 2018). Parasites can cause chronic inflammation and may result in AoI (Glinz et al. 2015). Helminths cause malabsorption of nutrients and diarrhea (Genta 1993). Other gastrointestinal diseases, such as gastroenteritis—acute or chronic inflammation (Merriman 2014)—likely caused issues for people in the past but are not associated with PCLs in other studies (e.g., Gomes et al. 2022; O'Donnell, Hill, et al. 2020).

Malnutrition and gastrointestinal infections may contribute to diarrheal diseases, which are a significant contributor to morbidity and mortality in children under five years of age today and likely shaped patterns of growth and mortality risk in Ancestral Puebloan populations (Schillaci et al. 2011). Gastroenteritis can quickly lead to dehydration and death; several routes to infection exist, including bacteria and parasites, but rotavirus is the most common agent (Siqueira et al. 2010). Because of the rapidity with which gastroenteritis kills, it is unlikely that it causes PCLs, given that prolonged illness seems to be a prerequisite (O'Donnell et al. 2023).

However, *H. pylori* infection often occurs in early childhood and was likely present in the Americas before Spanish arrival (Darling and Donoghue 2014). Hosts may experience chronic inflammation in the intestinal tract and reductions of micronutrient bioavailability over extended durations, which can result in iron loss and stunted growth (Muhsen and Cohen 2008). Although parasitism could possibly lead to PCL formation, partly because of its prolonged nature, this would need to be directly tested in living subjects.

A Note on Development

Cole and Waldron (2019) suggested that "simple porosity" of the orbit may represent normal developmental variation. Here, the probability of having PCLs drops with increasing age (Figure 4). Perhaps, due partly to developmental factors, PCLs are more likely to form in the young (see also Brickley 2018). Research findings may also indicate the resilience or robusticity of individuals who either never developed lesions or survived long enough for them to fully remodel.

Type 2 CO is significantly associated with younger ages at death (Figure 5), an association not noted for the vault bones. This relationship underscores the importance of recording and considering CO and PH morphology and location *before* diagnosing the presence of any disease. If Type 2 CO is *not* pathological, it should not be included in *any* analyses of past health and certainly should not be considered diagnostic of "anemia."

Conversely, when Type 2 CO is found alongside other PCL types, it may indicate pathological origins. In our study, two individuals with probable scurvy had Type 2 CO alongside Types 3 and 4. It would be prudent in any study of PCLs to examine whether there is an association between CO characterized *only by* fine, scattered foramina and young age in the pediatric population and what that association might mean.

Recommendations for Researchers

PCLs should be analyzed without assuming that they were caused by anemia or nutrient deficiency. Likewise, CO and PH should be considered separately, because they may have disassociated etiologies.

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When possible, PCLs should be evaluated within the context of (1) the entirety of an individual's skeleton; (2) specific PCL morphology; (3) age at PCL formation; that is, because they develop in early childhood, blood loss from menstruation or pregnancy resulting in iron deficiency anemia is unlikely; (4) the geographic and temporal location of the population; and (5) the actual, rather than the assumed, diet. If this evaluation is not possible, PCLs can be used as indicators of stress events of unknown origin.

Researchers need not eliminate use of the indicator—the presence or absence of PCLs—in analysis, but caution should be exercised when this approach is taken. As demonstrated here, this approach has utility. One definite benefit is the increased sample size, but a drawback could be the homogenization of potential causes; that is, all PCLs become signals of "anemia." If only PCL presence/absence is recorded, that data should not be used as equivocal evidence that PCLs signal nutrient deficiency, anemia, or any other condition. The best practice researchers can employ today is setting aside assumptions as to cause. PCLs should be treated as nonspecific indicators of stress until a complete differential diagnosis and analysis of the entire skeleton are undertaken (Grauer 2019:515) or the individual's medical history is known.

Literature that ascribes PCLs to anemia—or to any single cause, for that matter—is still useful for understanding the lived experience of past peoples. However, unless PCL morphology—diploic expansion in concert with pitting/porosity—is used to assess lesion etiology, no diagnosis can be made. PCLs have varied etiologies, and even when they manifest together in a single individual, they may not have the same causes.

Conclusions

Although PCLs are often associated with nutritional anemia, including that caused by iron deficiency, this relationship is probably only tangential to the primary insult. Anemia is a symptom of an underlying disease or disorder with many potential etiological drivers (see Grauer 2019:Figure 14.20). The problematic implications of associating CO and PH with nutritional anemia, despite their diverse etiologies, have been noted by multiple researchers (Cole and Waldron 2019; Grauer 2019; Mays 2012; Walker et al. 2009; Wapler et al. 2004), but the association continues to be made. Narrowing our lens to just iron deficiency or another specific form of nutritional anemia is an even more reductive approach. Instead of assuming anemia is the source, researchers should set aside preconceived notions of cause.

The cause(s) of PCLs is certainly open for debate in the Southwest—and likely elsewhere—especially when dietary data are considered. Surely, a proportion of PCLs are caused by nutritional anemia. However, human diets vary, even when people lean heavily on a staple grain. Ancestral Puebloans supplemented a maize-intensive diet with hunted and gathered foods. Many of those foods are rich in micronutrients, and some increase iron uptake and ferritin synthesis. So, it is unlikely that nutrient deficiency (iron-deficiency anemia, vitamin B_{12}), especially due to a maize-reliant diet, is the cause of most PCLs seen in the Southwest. Findings might differ in studies of different geographic locales or groups with different lifeways. Still, it is advisable to consider archaeological evidence for diet and cultural conditions alongside PCL morphology and frequency when assessing potential causative factors.

Supplemental Material. For supplemental material accompanying this article, visit https://doi.org/10.1017/aaq.2024.61.

Supplemental Table 1. Definitions of terminology and different types of anemia.

Supplemental Table 2. Number of coprolites by food type and site; for all references see Table 3; frequencies presented in Figure 4 were estimated using these totals. This data was used in Supplemental Figure 1 and Figure 2 (in this article); for the most part pollen studies required >=200 grains.

Supplemental Table 3. Sites where individuals included in Figure 5 are from. Number of individuals scored for cribra orbitalia (CO) and porotic hyperostosis (PH) here are presented for studies who reported PCLs for children (less than 15 years of age). This table also provides a range for the number of coprolites per site and study, but see Supplemental Table 2 for more detailed sample sizes of coprolites by site and food item. These data are not exhaustive.

Supplemental Figure 1. Frequency of coprolites with pollen/macrofossils by site.

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References Cited

Adams, Karen R. 2008. Subsistence and Plant Use during the Chacoan and Secondary Occupations at Salmon Ruin. In *Chaco's Northern Prodigies: Salmon, Aztec, and the Ascendancy of the Middle San Juan Region after AD 1100*, edited by Paul F. Reed, pp. 65–85. University of Utah Press, Salt Lake City.

Agarwal, K. N., N. Dhar, M. M. Shah, and O. P. Bhardwaj. 1970. Roentgenologic Changes in Iron Deficiency Anemia. *American Journal of Roentgenology* 110(3):635–637.

Ajmera, Rachael. 2018. Amaranth: An Ancient Grain with Impressive Health Benefits. *Healthline*, January 6. https://www.healthline.com/nutrition/amaranth-health-benefits, accessed December 18, 2019.

Akins, Nancy J. 1987. Human Remains from Pueblo Alto. In *Investigations at the Pueblo Alto Complex, Chaco Canyon 1975–1979: Volume III, Part 2*, edited by Frances Joan Mathien and Thomas C. Windes, pp. 789–792. National Park Service, US Department of the Interior, Santa Fe, New Mexico.

Aksoy, Muzaffer, Necdet Çamli, and Sakir Erdem. 1966. Roentgenographic Bone Changes in Chronic Iron Deficiency Anemia: A Study in Twelve Patients. *Blood* 27(5):677–686.

AlQahtani, Sakher J., Mark P. Hector, and Helen M. Liversidge. 2010. Brief Communication: The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology* 142(3):481–490.

Anderson, Amy S. 2022. Skeletal Indicators of Early Life Stress: Insights into Cribra Orbitalia and Porotic Hyperostosis in a Living Subsistence Population. PhD dissertation, Department of Anthropology, University of California, Santa Barbara.

Anschuetz, Kurt F. 2006. Tewa Fields, Tewa Traditions. In *Canyon Gardens: The Ancient Pueblo Landscapes of the American Southwest*, edited by V. B. Price and Baker H. Morrow, pp. 57–73. University of New Mexico Press, Albuquerque.

Badenhorst, Shaw, Jonathan C. Driver, and Susan C. Ryan. 2019. Desirable Meat: The Social Context of Meat Procurement at Albert Porter Pueblo, a Great House Community in the Central Mesa Verde Region. *Kiva* 85(3):191–213.

Baker, Brenda J., Tosha L. Dupras, and Matthew W. Tocheri. 2005. *The Osteology of Infants and Children*. Texas A&M University Anthropology Series No. 12. Texas A&M University Press, College Station.

Barriada-Bernal, L. Gerardo, L. Victoria Aquino-González, L. Leticia Méndez-Lagunas, Juan Rodríguez-Ramírez, and Sadoth Sandoval-Torres. 2018. Physical and Nutritional Characterization of Yucca Fruits (*Yucca mixtecana*). *Agrociencia* 52(3): 347–359.

Battillo, Jenna M. 2017. Supplementing Maize Agriculture in Basketmaker II Subsistence: Dietary Analysis of Human Paleofeces from Turkey Pen Ruin (42SA3714). PhD dissertation, Department of Anthropology, Southern Methodist University, Dallas, Texas.

Battillo, Jenna M. 2018. The Role of Corn Fungus in Basketmaker II Diet: A Paleonutrition Perspective on Early Corn Farming Adaptations. *Journal of Archaeological Science: Reports* 21:64–70.

Battillo, Jenna M. 2019. Farmers Who Forage: Interpreting Paleofecal Evidence of Wild Resource Use by Early Corn Farmers in the North American Southwest. *Archaeological and Anthropological Sciences* 11(11):5999–6016.

Beatrice, Jared S., and Angela Soler. 2016. Skeletal Indicators of Stress: A Component of the Biocultural Profile of Undocumented Migrants in Southern Arizona. *Journal of Forensic Sciences* 61(5):1164–1172.

Beatrice, Jared S., Angela Soler, Robin C. Reineke, and Daniel E. Martínez. 2021. Skeletal Evidence of Structural Violence among Undocumented Migrants from Mexico and Central America. *American Journal of Physical Anthropology* 176(4):584–605.

Beck, Margaret. 2001. Archaeological Signatures of Corn Preparation in the U.S. Southwest. *Kiva* 67(2):187–218.

Benson, Larry, Kenneth Petersen, and John Stein. 2007. Anasazi (Pre-Columbian Native-American) Migrations during the Middle-12th and Late-13th Centuries—Were they Drought Induced? *Climatic Change* 83(1–2):187–213.

Berry, David Richard. 1984. Disease and Climatological Relationship among Pueblo III-Pueblo IV Anasazi of the Colorado Plateau. PhD disssertation, Department of Anthropology, University of California, Los Angeles.

Beutler, Ernest. 1988. The Common Anemias. *JAMA* 259(16):2433–2437.

Brahmbhatt, Parthiv, Srajan Dashore, Manoj Dholu, and C. Raychaudhuri. 2017. Evaluation of Musculoskeletal Manifestations of Hemoglobinopathies by Conventional Imaging. *Journal of Integrated Health Sciences* 5(2):53–57.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

Brand, Michael James. 1994. Prehistoric Anasazi Diet: A Synthesis of Archaeological Evidence. Master's thesis, Department of Anthropology and Sociology, University of British Columbia, Vancouver, Canada.

Brickley, Megan B. 2018. Cribra Orbitalia and Porotic Hyperostosis: A Biological Approach to Diagnosis. *American Journal of Physical Anthropology* 167(4):896–902.

Brickley, Megan B. 2024. Perspectives on Anemia: Factors Confounding Understanding of Past Occurrence. *International Journal of Paleopathology* 44:90–104.

Brickley, Megan B., Rachel Ives, and Simon E. Mays. 2020. *The Bioarchaeology of Metabolic Bone Disease*. 2nd ed. Academic Press, Oxford.

Brickley, Megan B., and Brianne Morgan. 2023. Metabolic and Endocrine Diseases. In *The Routledge Handbook of Paleopathology*, edited by Anne L. Grauer, pp. 338–359. Routledge, London.

Buikstra, Jane E., and Douglas H. Ubelaker. 1994. *Standards for Data Collection from Human Skeletal Remains*. Research Series No. 44. Arkansas Archeological Survey, Fayetteville.

Cadwallader, Lauren, David G. Beresford-Jones, Oliver Q. Whaley, and Tamsin C. O'Connell. 2012. The Signs of Maize? A Reconsideration of What $\delta^{13}C$ Values Say about Palaeodiet in the Andean Region. *Human Ecology* 40(4):487–509.

Carlson, David S., George J. Armelagos, and Dennis P. Van Gerven. 1974. Factors Influencing the Etiology of Cribra Orbitalia in Prehistoric Nubia. *Journal of Human Evolution* 3(5):405–410.

Chaichun, Amnart, Laphatrada Yurasakpong, Athikhun Suwannakhan, Sitthichai Iamsaard, Supatcharee Arun, and Arada Chaiyamoon. 2021. Gross and Radiographic Appearance of Porotic Hyperostosis and Cribra Orbitalia in Thalassemia Affected Skulls. *Anatomy and Cell Biology* 54(2):280–284.

Clary, Karen H. 1983. Prehistoric Coprolite Remains from Chaco Canyon, New Mexico: Inferences for Anasazi Diet and Subsistence. Master's thesis, Department of Anthropology, University of New Mexico, Albuquerque.

Clary, Karen H. 1987. Coprolites from Pueblo Alto. In *Investigations at the Pueblo Alto Complex, Chaco Canyon, New Mexico 1975–1979: Volume III Part 1 Artifactual and Biological Analyses*, edited by Frances Joan Mathien & Thomas C. Windes, pp. 785–788. Publications in Archaeology No. 18F. National Park Service, US Department of the Interior, Santa Fe, New Mexico.

Cole, Garrard, and Tony Waldron. 2019. Cribra Orbitalia: Dissecting an Ill-Defined Phenomenon. *International Journal of Osteoarchaeology* 29(4):613–621.

Coltrain, Joan Brenner, and Joel C. Janetski. 2013. The Stable and Radio-Isotope Chemistry of Southeastern Utah Basketmaker II Burials: Dietary Analysis Using the Linear Mixing Model SISUS, Age and Sex Patterning, Geolocation and Temporal Patterning. *Journal of Archaeological Science* 40(12):4711–4730.

Cooley, Thomas B., and Pearl Lee. 1925. A Series of Cases of Splenomegaly in Children, with Anemia and Peculiar Bone Changes. *Transactions of the American Pediatric Society* 37:29–30.

Cordell, Linda S. 1995. Tracing Migration Pathways from the Receiving End. *Journal of Anthropological Archaeology* 14(2):203–211.

Crandall, John J. 2014. Scurvy in the Greater American Southwest: Modeling Micronutrition and Biosocial Processes in Contexts of Resource Stress. *International Journal of Paleopathology* 5:46–54.

Crown, Patricia L., Janet D. Orcutt, and Timothy A. Kohler. 1996. Pueblo Cultures in Transition: The Northern Rio Grande. In *The Prehistoric Pueblo World, A.D. 1150–1350*, edited by Michael A. Adler, pp. 188–204. University of Arizona Press, Tucson.

Cummings, Linda S., Chad Yost, Kathryn Puseman, and Melissa K. Logan. 2009. *Pollen, Starch, Parasite, Phytolith, Macrofloral, and Organic Residue (FTIR) Analysis of Coprolites From Room 225, West Ruin, Aztec Ruins National Monument, Aztec, New Mexico*. Technical Report 09-86. Paleo Research Institute, Golden, Colorado.

Danforth, Marie Elaine, Della Collins Cook, and Stanley G. Knick III. 1994. The Human Remains from Carter Ranch Pueblo, Arizona: Health in Isolation. *American Antiquity* 59(1):88–101.

Darling, Millie I., and Helen D. Donoghue. 2014. Insights from Paleomicrobiology into the Indigenous Peoples of Pre-Colonial America—A Review. *Memórias do Instituto Oswaldo Cruz* 109(2):131–139.

Diggs, L. W., H. N. Pulliam, and J. C. King. 1937. The Bone Changes in Sickle Cell Anemia. *Southern Medical Journal* 30(3):249–259.

Doebley, John F., and Vorsila L. Bohrer. 1983. Maize Variability and Cultural Selection at Salmon Ruin, New Mexico. *Kiva* 49(1–2):19–37.

Dombrosky, Jonathan, Alexi C. Besser, Emma A. Elliott Smith, Cyler Conrad, Laura Pagès Barceló, and Seth D. Newsome. 2020. Resource Risk and Stability in the Zooarchaeological Record: The Case of Pueblo Fishing in the Middle Rio Grande, New Mexico. *Archaeological and Anthropological Sciences* 12(10):248.

Durand, Kathy Roler, and Stephen R. Durand. 2008. Animal Bone from Salmon Ruins and Other Great Houses: Faunal Exploitation in the Chaco World. In *Chaco's Northern Prodigies: Salmon, Aztec, and the Ascendancy of the Middle San Juan Region after AD 1100*, edited by Paul F. Reed, pp. 96–112. University of Utah Press, Salt Lake City.

Duya, C. O., Mildred P. Nawiri, and Alphonse W. Wafula. 2018. Beta-Carotene Levels Increase with Leaf Maturity of Amaranthus hybridus (L) Grown in Different Soil-Types of Kwale County, Kenya. *EC Nutrition* 13(5):299–304.

East, Anna Louise. 2008. Reproduction and Prenatal Care in Arizona Prehistory: An Examination of Patterns of Health in Perinates and Children at Grasshopper, Point of Pines, and Turkey Creek Pueblos. PhD dissertation, Department of Anthropology, University of New Mexico, Albuquerque.

Eggan, Dorothy. 1943. The General Problem of Hopi Adjustment. *American Anthropologist* 45(3):357–373.

Eggan, Fred. 1950. *Social Organization of the Western Pueblos*. University of Chicago Press, Chicago.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

El-Najjar, Mahmoud Y. 1986. The Biology and Health of the Prehistoric Inhabitants of Canyon de Chelly. In *Archaeological Investigations at Antelope House*, edited by Don P. Morris, pp. 206–220. National Park Service, US Department of the Interior, Washington, DC.

El-Najjar, Mahmoud Y., Betsy Lozoff, and Dennis J. Ryan. 1975. The Paleoepidemiology of Porotic Hyperostosis in the American Southwest: Radiological and Ecological Considerations. *American Journal of Roentgenology* 125(4):918–924.

El-Najjar, Mahmoud Y., Dennis J. Ryan, Christy G. Turner, and Betsy Lozoff. 1976. The Etiology of Porotic Hyperostosis among the Prehistoric and Historic Anasazi Indians of the Southwestern United States. *American Journal of Physical Anthropology* 44(3):477–488.

Eng, Lie-Injo Luan. 1958. Chronic Iron Deficiency Anaemia with Bone Changes Resembling Cooley's Anaemia. *Acta Haematologica* 19(4–5):263–268.

Epsley, Scott, Samuel Tadros, Alexander Farid, Daniel Kargilis, Sameer Mehta, and Chamith S. Rajapakse. 2020. The Effect of Inflammation on Bone. *Frontiers in Physiology* 11:511799.

Ezzo, Joseph A. 1994. Paleonutrition at Grasshopper Pueblo. In *Paleonutrition: The Diet and Health of Prehistoric Americans*, edited by Kristin D. Sobolik, pp. 265–279. Center for Archaeological Investigations Occasional Paper No. 22. Southern Illinois University, Carbondale.

Ferguson, Cheryl. 1980. Analysis of Human Remains. In *Tijeras Canyon: Analyses of the Past*, edited by Linda S. Cordell, pp. 121–148. University of New Mexico Press, Albuquerque.

Fink, T. Michael. 1985. Tuberculosis and Anemia in a Pueblo II–III (ca. AD 900–1300) Anasazi Child from New Mexico. In *Health and Disease in the Prehistoric Southwest*, Anthropological Research Papers Vol. 34, edited by Charles F. Merbs and Robert J. Miller, pp. 359–379. Arizona State University, Tempe.

Fry, Gary F., and H. Johnson Hall. 1986. Human Coprolites. In *Archaeological Investigations at Antelope House*, edited by Don P. Morris, pp. 165–188. National Park Service, US Department of the Interior, Washington, DC.

Ganz, Tomas. 2019. Anemia of Inflammation. *New England Journal of Medicine* 381(12):1148–1157.

García-Casal, Maria N. 2006. Carotenoids Increase Iron Absorption from Cereal-Based Food in the Human. *Nutrition Research* 26(7):340–344.

Genta, Robert M. 1993. Diarrhea in Helminthic Infections. *Clinical Infectious Diseases* 16(S2):S122–S129.

Gillespie, William B. 1981. *Summary of Osteological Remains from Chaco Coprolites*. Submitted to Chaco Center, National Park Service, University of New Mexico, Albuquerque.

Glinz, Dominik, Richard F. Hurrell, Aurélie A. Righetti, Christophe Zeder, Lukas G. Adiossan, Harold Tjalsma, Jürg Utzinger, Michael B. Zimmermann, Eliézer K. N'Goran, and Rita Wegmüller. 2015. In Ivorian School-Age Children, Infection with Hookworm Does Not Reduce Dietary Iron Absorption or Systemic Iron Utilization, Whereas Afebrile Plasmodium Falciparum Infection Reduces Iron Absorption by Half. *American Journal of Clinical Nutrition* 101(3):462–470.

Gomes, Ricardo A. M. P., Jimmy Petit, Olivier Dutour, and Ana Luisa Santos. 2022. Frequency and Co-Occurrence of Porous Skeletal Lesions in Identified Non-Adults from Portugal (19th to 20th Centuries) and Its Association with Respiratory Infections as Cause of Death. *International Journal of Osteoarchaeology* 32(5):1061–1072.

Gonzalez, Nancie L. 1974. Changing Dietary Patterns of North American Indians. In *National Nutrition Policy: Nutrition and Special Groups, A Working Paper*, edited by Freeman H. Quimby and Cynthia B. Chapman, pp. 50–68. US Government Printing Office, Washington, DC.

Grauer, Anne L. 2019. Circulatory, Reticuloendothelial, and Hematopoietic Disorders. In *Ortner's Identification of Pathological Conditions in Human Skeletal Remains*, 3rd ed., edited by Jane E. Buikstra, pp. 491–529. Academic Press, London.

Haines, Christopher J., Aun Woon Soon, and Danielle Mercurio. 2013. Community-Acquired Pneumonia in Pediatric Populations. *Emergency Medicine Reports* 34(17):197–208.

Hannah, Scott S., Sonyia McFadden, Andrea McNeilly, and Conor McClean. 2021. "Take My Bone Away?" Hypoxia and Bone: A Narrative Review. *Journal of Cellular Physiology* 236(2):721–740.

Hard, Robert J., Raymond P. Mauldin, and Gerry R. Raymond. 1996. Mano Size, Stable Carbon Isotope Ratios, and Macrobotanical Remains as Multiple Lines of Evidence of Maize Dependence in the American Southwest. *Journal of Archaeological Method and Theory* 3(3):253–318.

Harrod, Ryan P. 2012. Centers of Control: Revealing Elites among the Ancestral Pueblo during the "Chaco Phenomenon." *International Journal of Paleopathology* 2(2–3):123–135.

Hens, Samantha M., Kanya Godde, and Kristin M. Macak. 2019. Iron Deficiency Anemia, Population Health and Frailty in a Modern Portuguese Skeletal Sample. *PLoS ONE* 14(3):e0213369.

Holliday, Diane Young. 1996. Were Some More Equal? Diet and Health at the NAN Ranch Pueblo, Mimbres Valley, New Mexico. PhD dissertation, Department of Anthropology, University of Wisconsin, Madison.

Huckell, Lisa W., and Mollie S. Toll. 2004. Wild Plant Use in the North American Southwest. In *People and Plants in Ancient Western North America*, edited by Paul E. Minnis, pp. 37–114. Smithsonian Institution, Washington, DC.

Huss-Ashmore, Rebecca, Alan H. Goodman, and George J. Armelagos. 1982. Nutritional Inference from Paleopathology. In *Advances in Archaeological Method and Theory*, Vol. 5, edited by Michael B. Schiffer, pp. 395–474. Academic Press, New York.

Hutchinson, Dale L., Lynette Norr, and Mark F. Teaford. 2007. Outer Coast Foragers and Inner Coast Farmers in Late Prehistoric North Carolina. In *Ancient Health: Skeletal Indicators of Agricultural and Economic Intensification*, edited by Mark Nathan Cohen and Gillian Margaret Mountford Crane-Kramer, pp. 52–64. University Press of Florida, Gainesville.

Ives, Rachel. 2018. Rare Paleopathological Insights into Vitamin D Deficiency Rickets, Co-Occurring Illnesses, and Documented Cause of Death in Mid-19th Century London, UK. *International Journal of Paleopathology* 23:76–87.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

Jann, Ben. 2013. COEFPLOT: Stata Module to Plot Regression Coefficients and Other Results. StataCorp, College Station, Texas.
Jatautis, Šarūnas, Ieva Mitokaitė, and Rimantas Jankauskas. 2011. Analysis of Cribra Orbitalia in the Earliest Inhabitants of Medieval Vilnius. *Anthropological Review* 74:57–68.
Jorgensen, Julia. 1975. A Room Use Analysis of Table Rock Pueblo, Arizona. *Journal of Anthropological Research* 31(2):149–161.
Jurado, Rafael L. 1997. Iron, Infections, and Anemia of Inflammation. *Clinical Infectious Diseases* 25(4):888–895.
Keenleyside, A., and K. Panayotova. 2006. Cribra Orbitalia and Porotic Hyperostosis in a Greek Colonial Population (5th to 3rd Centuries BC) from the Black Sea. *International Journal of Osteoarchaeology* 16(5):373–384.
Kellner, Corina M., and Margaret J. Schoeninger. 2007. A Simple Carbon Isotope Model for Reconstructing Prehistoric Human Diet. *American Journal of Physical Anthropology* 133(4):1112–1127.
Kent, Susan. 1986. The Influence of Sedentism and Aggregation on Porotic Hyperostosis and Anaemia: A Case Study. *Man* 21(4): 605–636.
Kent, Susan. 2000. Iron Deficiency and Anemia of Chronic Disease. In *Cambridge World History of Food*, Vol. 1, edited by Kenneth F. Kiple and Kriemhild Conee Ornelas, pp. 919–939. Cambridge University Press, Cambridge.
Kinder, David H., Karen R. Adams, and Harry J. Wilson. 2017. *Solanum jamesii*: Evidence for Cultivation of Wild Potato Tubers by Ancestral Puebloan Groups. *Journal of Ethnobiology* 37(2):218–241.
Kohler, Timothy A., Mark D. Varien, and Aaron M. Wright (editors). 2010. *Leaving Mesa Verde: Peril and Change in the Thirteenth-Century Southwest*. University of Arizona Press, Tucson.
Kuckelman, Kristin A., and Debra L. Martin. 2007. Human Skeletal Remains. In *The Archaeology of Sand Canyon Pueblo: Intensive Excavations at a Late-Thirteenth-Century Village in Southwestern Colorado*, edited by Kristin A. Kuckelman. Crow Canyon Archaeological Center, Cortez, Colorado. Electronic document, https://crowcanyon.org/ResearchReports/SandCanyon/Text/scpw_human skeletalremains, accessed February 5, 2025.
Kunitz, Stephen J. 1970. Disease and Death among the Anasazi: Some Notes on Southwestern Paleoepidemiology. *El Palacio* 76(3):17–22.
Kunitz, Stephen J., and Robert C. Euler. 1972. *Aspects of Southwestern Paleoepidemiology*. Anthropological Reports No. 2. Prescott College Press, Prescott, Arizona.
Lallo, John W., George J. Armelagos, and Robert P. Mensforth. 1977. The Role of Diet, Disease, and Physiology in the Origin of Porotic Hyperostosis. *Human Biology* 49(3):471–483.
Lambert, Patricia M. 2002. Rib Lesions in a Prehistoric Puebloan Sample from Southwestern Colorado. *American Journal of Physical Anthropology* 117(4):281–292.
Lanzkowsky, Philip. 1968. Radiological Features of Iron-Deficiency Anemia. *American Journal of Diseases of Children* 116(1):16–29.
Layrisse, Miguel, Carlos Martinez-Torres, and Marcel Roche. 1968. Effect of Interaction of Various Foods on Iron Absorption. *American Journal of Clinical Nutrition* 21(10):1175–1183.
Levy, Jerrold E. 1992. *Orayvi Revisited: Social Stratification in an "Egalitarian" Society*. School of American Research, Santa Fe, New Mexico.
Lewis, Mary E. 2012. Thalassaemia: Its Diagnosis and Interpretation in Past Skeletal Populations. *International Journal of Osteoarchaeology* 22(6):685–693.
Lewis, Mary E. 2017. *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults*. Academic Press, Elsevier, London.
Lindaman, Lynn M. 2001. Bone Healing in Children. *Clinics in Podiatric Medicine and Surgery* 18(1):97–108.
Marden, Kerriann. 2011. Taphonomy, Paleopathology, and Mortuary Variability in Chaco Canyon: Using Bioarchaeological and Forensic Methods to Understand Ancient Cultural Practices. PhD dissertation, Department of Anthropology, Tulane University, New Orleans, Louisiana.
Martin, Debra L., and Anna J. Osterholtz. 2016. Broken Bodies and Broken Bones: Biocultural Approaches to Ancient Slavery and Torture. In *New Directions in Biocultural Anthropology*, edited by Molly K. Zuckerman and Debra L. Martin, pp. 471–490. John Wiley & Sons, Hoboken, New Jersey.
Martinson, Elizabeth Ann. 2002. Reassessing the Etiology of Cribra Orbitalia and Porotic Hyperostosis: A Case Study of the Chiribaya of the Osmore Drainage, Perú. PhD dissertation, Department of Anthropology, University of New Mexico, Albuquerque.
Mays, Simon E. 2008. A Likely Case of Scurvy from Early Bronze Age Britain. *International Journal of Osteoarchaeology* 18(2): 178–187.
Mays, Simon E. 2012. The Relationship between Paleopathology and the Clinical Sciences. In *A Companion to Paleopathology*, edited by Anne L. Grauer, pp. 285–309. Wiley-Blackwell, Chichester, UK.
Mays, Simon E. 2018a. How Should We Diagnose Disease in Palaeopathology? Some Epistemological Considerations. *International Journal of Paleopathology* 20:12–19.
Mays, Simon E. 2018b. Micronutrient Deficiency Diseases: Anemia, Scurvy, and Rickets. In *The International Encyclopedia of Biological Anthropology*, Vol. 1, edited by Wenda Trevathan, Matt Cartmill, Darna L. Dufour, Clark Spencer Larsen, Dennis H. O'Rourke, Karen Rosenberg, and Karen B. Strier. John Wiley & Sons, Hoboken, New Jersey. https://doi.org/10.1002/9781118584538.ieba0271.
McIlvaine, Britney Kyle. 2015. Implications of Reappraising the Iron-Deficiency Anemia Hypothesis. *International Journal of Osteoarchaeology* 25(6):997–1000.
McKusick, Charmion R. 1982. Avifauna from Grasshopper Pueblo. In *Multidisciplinary Research at Grasshopper Pueblo, Arizona*, edited by William A. Longacre, Sally J. Holbrook, and Michael W. Graves, Anthropological Papers of the University of Arizona No. 40, pp. 87–96. University of Arizona Press, Tucson.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

McPherson, Cait B. 2021. Examining Developmental Plasticity in the Skeletal System through a Sensitive Developmental Windows Framework. *American Journal of Physical Anthropology* 176(2):163–178.

Merbs, Charles F., and Robert J. Miller. (editors). 1985. *Health and Disease in the Prehistoric Southwest*. Anthropological Research Papers No. 34. Arizona State University, Tempe.

Merriman, Harold. 2014. Infectious Diseases. In *Acute Care Handbook for Physical Therapists*, 4th ed., edited by Jaime C. Paz and Michele P. West, pp. 313–334. Saunders, St. Louis, Missouri.

Minnis, Paul E. 1989. Prehistoric Diet in the Northern Southwest: Macroplant Remains from Four Corners Feces. *American Antiquity* 54(3):543–563. https://doi.org/10.2307/280782.

Moerman, Daniel E. 2010. *Native American Food Plants: An Ethnobotanical Dictionary*. Timber Press, Portland, Oregon.

Moore, C. V. 1968. Iron. In *Modern Nutrition in Health and Disease*, 4th ed., edited by Michael G. Wohl and Robert S. Goodhart, pp. 213–227. Lea and Febiger, Philadelphia.

Moseley, John E. 1965. The Paleopathologic Riddle of "Symmetrical Osteoporosis." *American Journal of Roentgenology* 95(1): 135–142.

Mtvarelidze, Z. G., A. N. Kvezereli-Kopadze, and M. A. Kvezereli-Kopadze. 2009. Megaloblastic-Vitamin B12 Deficiency Anemia in Childhood. *Georgian Medical News* 170(5):57–60.

Muhsen, Khitam, and Dani Cohen. 2008. Helicobacter pylori Infection and Iron Stores: A Systematic Review and Meta-Analysis. *Helicobacter* 13(5):323–340.

Mulholland, Kim. 2007. Perspectives on the Burden of Pneumonia in Children. *Vaccine* 25(13):2394–2397.

Nayak, Lalitha, Lawrence B. Gardner, and Jane A. Little. 2018. Anemia of Chronic Diseases. In *Hematology: Basic Principles and Practice*, 7th ed., edited by Ronald Hoffman, Edward J. Benz Jr., Leslie E. Silberstein, Helen E. Heslop, Jeffrey I. Weitz, John Anastasi, Mohamed E. Salama, and Syed Ali Abutalib, pp. 491–496. Elsevier, Philadelphia.

Nistor, Eleonora, Vasileios A. Bampidis, Nicolae Păcală, Marius Pentea, J. Tozer, and H. Prundeanu. 2013. Nutrient Content of Rabbit Meat as Compared to Chicken, Beef and Pork Meat. *Journal of Animal Production Advances* 3(4):172–176.

Obertová, Zuzana, and Milan Thurzo. 2004. Cribra Orbitalia as an Indicator of Stress in the Early Medieval Slavic Population from Borovce (Slovakia). *Anthropologie* 42(2):189–194.

O'Donnell, Lexi. 2019. Indicators of Stress and Their Association with Frailty in the Precontact Southwestern United States. *American Journal of Physical Anthropology* 170(3):404–417.

O'Donnell, Lexi [Alexis], Shamsi Daneshvari Berry, and Heather J. H. Edgar. 2017. Can Cephalometrics Discriminate between the Sexes in a Diverse Juvenile Sample? *Journal of Forensic Sciences* 62(3):735–740.

O'Donnell, Lexi, Jane E. Buikstra, Ethan C. Hill, Amy S. Anderson, and Michael J. O'Donnell Jr. 2023. Skeletal Manifestations of Disease Experience: Length of Illness and Porous Cranial Lesion Formation in a Contemporary Juvenile Mortality Sample. *American Journal of Human Biology* 35(8):e23896.

O'Donnell, Lexi, John J. Green, Ethan C. Hill, and Michael J. O'Donnell Jr. 2024. Biocultural and Social Determinants of Ill Health and Early Mortality in a New Mexican Paediatric Autopsy Sample. *Journal of Biosocial Science* 56(4):693–714.

O'Donnell, Lexi, Ethan C. Hill, Amy S. Anderson, and Heather J. H. Edgar. 2020. Cribra Orbitalia and Porotic Hyperostosis Are Associated with Respiratory Infections in a Contemporary Mortality Sample from New Mexico. *American Journal of Physical Anthropology* 173(4):721–733.

O'Donnell, Lexi, Ethan C. Hill, Amy S. Anderson, and Heather J. H. Edgar. 2022. A Biological Approach to Adult Sex Differences in Skeletal Indicators of Childhood Stress. *American Journal of Biological Anthropology* 177(3):381–401.

O'Donnell, Lexi, and Emily Moes. 2021. Sex Differences in Linear Enamel Hypoplasia Prevalence and Frailty in Ancestral Puebloans. *Journal of Archaeological Science: Reports* 39:103153.

O'Donnell, Lexi, Jana Valesca Meyer, and Corey S. Ragsdale. 2020. Trade Relationships and Gene Flow at Pottery Mound Pueblo, New Mexico. *American Antiquity* 85(3):492–515.

Ortman, Scott G. 2010. Evidence of a Mesa Verde Homeland for the Tewa Pueblos. In *Leaving Mesa Verde: Peril and Change in the Thirteenth-Century Southwest*, edited by Timothy A. Kohler, Mark D. Varien, and Aaron M. Wright, pp. 222–261. University of Arizona Press, Tucson.

Ortner, Donald J. 2003. *Identification of Pathological Disorders in Human Skeletal Remains*. 2nd ed. Academic Press, Amsterdam.

Ortner, Donald J. 2012. Differential Diagnosis and Issues in Disease Classification. In *A Companion to Paleopathology*, edited by Anne L. Grauer, pp. 250–267. Wiley-Blackwell, Chichester, UK.

Ortner, Donald J., Whitney Butler, Jessica Cafarella, and Lauren Milligan. 2001. Evidence of Probable Scurvy in Subadults from Archaeological Sites in North America. *American Journal of Physical Anthropology* 114(4):343–351.

Oxenham, Marc F., and Ivor Cavill. 2010. Porotic Hyperostosis and Cribra Orbitalia: The Erythropoietic Response to Iron-Deficiency Anaemia. *Anthropological Science* 118(3):199–200.

Palkovich, Ann M. 1980. *Pueblo Population and Society: The Arroyo Hondo Skeletal and Mortuary Remains*. School of American Research Press, Santa Fe, New Mexico.

Palkovich, Ann M. (1985). Interpreting prehistoric morbidity incidence and mortality risk: Nutritional stress at Arroyo Hondo pueblo, New Mexico. In Charles Merbs & R. Miller (Eds.), *Health and disease in the prehistoric Southwest* (pp. 139–164).

Paseka, Rachel E., Carrie C. Heitman, and Karl J. Reinhard. 2018. New Evidence of Ancient Parasitism among Late Archaic and Ancestral Puebloan Residents of Chaco Canyon. *Journal of Archaeological Science: Reports* 18:51–58.

Perry, Elizabeth M., Ann L. W. Stodder, and Charles A. Bollong (editors). 2010. *Animas-La Plata Project, Volume XV: Bioarchaeology*. Anthropological Research Paper No. 10. SWCA Environmental Consultants, Phoenix, Arizona.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

Rawlings, Tiffany A., and Jonathan C. Driver. 2010. Paleodiet of Domestic Turkey, Shields Pueblo (5MT3807), Colorado: Isotopic Analysis and Its Implications for Care of a Household Domesticate. *Journal of Archaeological Science* 37(10):2433–2441.

Reinhard, Karl J. 1988. Diet, Parasitism, and Anemia in the Prehistoric Southwest. PhD dissertation, Department of Anthropology, Texas A&M University, College Station.

Reinhard, Karl J. 1992. Patterns of Diet, Parasitism, and Anemia in Prehistoric West North America. In *Diet, Demography, and Disease: Changing Perspectives on Anemia*, edited by Patricia Stuart-Macadam and Susan Kent, pp. 219–258. Aldine de Gruyter, Hawthorne, New York.

Reinhard, Karl J. 2006. Coprolite Analysis from Salmon Ruins: the San Juan Occupation. In *Thirty-Five Years of Archaeological Research at Salmon Ruins, New Mexico: Volume Three: Archaeobotanical Research and Other Analytical Studies*, edited by Paul F. Reed, pp. 875–888. Center for Desert Archaeology, Salmon Ruins Museum, Tucson, Arizona.

Reinhard, Karl J. 2008a. Parasite Pathoecology of Salmon Pueblo and Other Chacoan Great Houses: The Healthiest and Wormiest Ancestral Puebloans. In *Chaco's Northern Prodigies: Salmon, Aztec, and the Ascendancy of the Middle San Juan Region after AD 1100*, edited by Paul F. Reed, pp. 86–95. University of Utah Press, Salt Lake City.

Reinhard, Karl J. 2008b. Pathoecology of Two Ancestral Pueblo Villages. In *Case Studies in Environmental Archaeology*, 2nd ed., edited by Elizabeth J. Reitz, C. Margaret Scarry, and Sylvia J. Scudder, pp. 191–209. Springer, New York.

Reinhard, Karl J., and Vaughn M. Bryant. 1992. Coprolite Analysis: A Biological Perspective on Archaeology. In *Archaeological Method and Theory*, Vol. 4, edited by Michael B. Schiffer, pp. 245–288. University of Arizona Press, Tucson

Reinhard, Karl J., and Dennis R. Danielson. 2005. Pervasiveness of Phytoliths in Prehistoric Southwestern Diet and Implications for Regional and Temporal Trends for Dental Microwear. *Journal of Archaeological Science* 32(7):981–988.

Reinhard, Karl J., and Richard H. Hevly. 1991. Dietary and Parasitological Analysis of Coprolites Recovered from Mummy 5, Ventana Cave, Arizona. *Kiva* 56(3):319–325.

Rivera, Frances, and Marta Mirazón Lahr. 2017. New Evidence Suggesting a Dissociated Etiology for Cribra Orbitalia and Porotic Hyperostosis. *American Journal of Physical Anthropology* 164(1):76–96.

Roberts, Charlotte A., and Jane E. Buikstra. 2019. Bacterial Infections. In *Ortner's Identification of Pathological Conditions in Human Skeletal Remains*, 3rd ed., edited by Jane E. Buikstra, pp. 321–439. Academic Press, London.

Roney, John R. 1995. Mesa Verdean Manifestations South of the San Juan River. *Journal of Anthropological Archaeology* 14(2):170–183.

Rothschild, Bruce M. 2012. Extirpolation of the Mythology That Porotic Hyperostosis Is Caused by Iron Deficiency Secondary to Dietary Shift to Maize. *Advances in Anthropology* 2(3):157–160.

RStudio Team. 2016. *RStudio: Integrated Development for R*. RStudio, Boston.

Schapkaitz, E., S. Buldeo, and J. N. Mahlangu. 2015. Diagnosis of Iron Deficiency Anaemia in Hospital Patients: Use of the Reticulocyte Haemoglobin Content to Differentiate Iron Deficiency Anaemia from Anaemia of Chronic Disease. *South African Medical Journal* 106(1):53–54.

Schats, Rachel. 2023. Developing an Archaeology of Malaria: A Critical Review of Current Approaches and a Discussion on Ways Forward. *International Journal of Paleopathology* 41:32–42.

Schattmann, Annabelle, Benoît Bertrand, Sophie Vatteoni, and Megan Brickley. 2016. Approaches to Co-occurrence: Scurvy and Rickets in Infants and Young Children of 16–18th Century Douai, France. *International Journal of Paleopathology* 12: 63–75.

Scheuer, Louise, and Sue M. Black. 2000. *Developmental Juvenile Osteology*. Academic Press, San Diego, California.

Scheuer, Louise, and Sue Black. 2004. *The Juvenile Skeleton*. 1st ed. Elsevier, London.

Schillaci, Michael A., and Steven A. Lakatos. 2016. Refiguring the Population History of the Tewa Basin. *Kiva* 82(4):364–386.

Schillaci, Michael A., Dejana Nikitovic, Nancy J. Akins, Lianne Tripp, and Ann M. Palkovich. 2011. Infant and Juvenile Growth in Ancestral Pueblo Indians. *American Journal of Physical Anthropology* 145(2):318–326.

Schlanger, Sarah H., and Richard H. Wilshusen. 1993. Local Abandonments and Regional Conditions in the North American Southwest. In *Abandonment of Settlements and Regions: Ethnoarchaeological and Archaeological Approaches*, edited by Catherine M. Cameron and Steve A. Tomka, pp. 85–98. Cambridge University Press, Cambridge.

Schultz, Michael. 2003. Light Microscopic Analysis in Skeletal Paleopathology. In *Identification of Pathological Conditions in Human Skeletal Remains*, 2nd ed., edited by Donald J. Ortner, pp. 73–108. Academic Press, Amsterdam.

Scott, Linda J. 1979. Dietary Inferences from Hoy House Coprolites: A Palynological Interpretation. *Kiva* 44(2–3):257–281.

Sebes, Jeno I., and L. W. Diggs. 1979. Radiographic Changes of the Skull in Sickle Cell Anemia. *American Journal of Roentgenology* 132(3):373–377.

Sheldon, Wilfrid. 1936. Anæmia with Bone Changes in the Skull. *Proceedings of the Royal Society of Medicine* 29(7):743.

Sheridan, Susan G., and Dennis P. Van Gerven. 1997. Female Biological Resiliency: Differential Stress Response by Sex in Human Remains from Ancient Nubia. *Human Evolution* 12(4):241–252.

Shine, James W. 1997. Microcytic Anemia. *American Family Physician* 55(7):2455–2462.

Shipman, Jeffrey H. 2006. A Brief Overview of Human Skeletal Remains From Salmon Ruins. In *Thirty-Five Years of Archaeological Research at Salmon Ruins, New Mexico: Volume One: Introduction, Architecture, Chronology, and Conclusions*, edited by Paul F. Reed, pp. 327–330. Center for Desert Archaeology, Salmon Ruins Museum, Tucson, Arizona.

Siqueira, Alessandra A., A. C. F. S. Santelli, L. R. Alencar Jr., M. P. Dantas, C. P. N. Dimech, G. M. I. Carmo, D. A. Santos, et al. 2010. Outreak of Acute Gastroenteritis in Young Children with Death due to Rotavirus Genotype G9 in Rio Branco, Brazilian Amazon Region, 2005. *International Journal of Infectious Diseases* 14(10):e898–e903.

Smith-Guzman, Nicole E. 2015. Cribra Orbitalia in the Ancient Nile Valley and Its Connection to Malaria. *International Journal of Paleopathology* 10:1–12.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

Snoddy, Anne Marie E., Hallie R. Buckley, Gail E. Elliott, Vivien G. Standen, Bernardo T. Arriaza, and Siân E. Halcrow. 2018. Macroscopic Features of Scurvy in Human Skeletal Remains: A Literature Synthesis and Diagnostic Guide. *American Journal of Physical Anthropology* 167(4):876–895.

Snow, Cordelia T. 2002. Fish Tales: The Use of Freshwater Fish in New Mexico from AD 1000 to 1900. In *Forward into the Past: Papers in Honor of Teddy Lou and Francis Stickney*, edited by Regge N. Wiseman, Thomas C. O'Laughlin, and Cordelia T. Snow, pp. 119–131. Archaeological Society of New Mexico, Albuquerque.

Snow, David H. 1990. Tener Comal y Metate: Protohistoric Rio Grande Maize Use and Diet. In *Perspectives on Southwestern Prehistory*, edited by Paul E. Minnis and Charles L. Redman, pp. 289–300. Westview Press, Boulder, Colorado.

Spivak, Jerry L. 2002. Iron and the Anemia of Chronic Disease. *Oncology* 16(9 S10):25–33.

Stallknecht, G. F., and J. R. Schulz-Schaeffer. 1993. Amaranth Rediscovered. In *New Crops*, edited by Jules Janick and James E. Simon, pp. 211–218. Wiley, New York.

StataCorp. 2017. *Stata Statistical Software: Release 15*. StataCorp, College Station, Texas.

Stearns, Stephen C. 1992. *The Evolution of Life Histories*. Oxford University Press, Oxford.

Stiger, Mark A. 1977. Anasazi Diet: The Coprolite Evidence. Master's thesis, Department of Anthropology, University of Colorado, Boulder.

Stiger, Mark A. 1979. Mesa Verde Subsistence Patterns from Basketmaker to Pueblo III. *Kiva* 44(2–3):133–144.

Stodder, Ann L. W. 1989. Bioarchaeological Resources Survey of the Basin and Range Region. In *Human Adaptations and Cultural Change in the Greater Southwest: An Overview of Archaeological Resources in the Basin and Range Province*, edited by Alan H. Simmons, Ann L. W. Stodder, Douglas D. Dykeman, and Patricia A. Hicks, pp. 141–190. Arkansas Archeological Survey, Fayetteville.

Stodder, Ann L. W. 1996. Paleoepidemiology of Eastern and Western Pueblo Communities in Protohistoric and Early Contact Period New Mexico. In *Bioarchaeology of Native American Adaptation in the Spanish Borderlands*, edited by Brenda J. Baker and Lisa Kealhofer, pp. 148–176. University of Florida Press, Gainesville.

Stodder, Ann L. W., and Debra L. Martin. 1992. Health and Disease in the Southwest before and after Spanish Contact. In *Disease and Demography in the Americas*, edited by John W. Verano and Douglas H. Ubelaker, pp. 55–73. Smithsonian Institution, Washington, DC.

Stodder, Ann L. W., Anna J. Osterholtz, Kathy Mowrer, and Erin Salisbury. 2010. Skeletal Pathology and Anomalies. In *Animas-La Plata Project, Volume XV: Bioarchaeology*, Anthropological Research Paper No. 10, edited by Elizabeth M. Perry, Ann L. W. Stodder, and Charles A. Bollong, pp. 89–156. SWCA Environmental Consultants, Phoenix, Arizona.

Stone, Katie L., Douglas C. Bauer, Deborah Sellmeyer, and Steven R. Cummings. 2004. Low Serum Vitamin B-12 Levels Are Associated with Increased Hip Bone Loss in Older Women: A Prospective Study. *Journal of Clinical Endocrinology & Metabolism* 89(3):1217–1221.

Stuart-Macadam, Patricia [Patty]. 1985. Porotic Hyperostosis: Representative of a Childhood Condition. *American Journal of Physical Anthropology* 66(4):391–398.

Stuart-Macadam, Patricia. 1991a. Anaemia in Roman Britain: Poundbury Camp. In *Health in Past Societies: Biocultural Interpretations of Human Skeletal Remains in Archaeological Contexts*, edited by Helen Bush and Marek Zvelebil, pp. 101–113. BAR International Series 567. British Archaeological Reports, Oxford.

Stuart-Macadam, Patricia. 1991b. Porotic Hyperostosis: Changing Interpretations. In *Human Paleopathology: Current Syntheses and Future Options*, edited by Donald J. Ortner and Arthur C. Aufderheide, pp. 36–39. Smithsonian Institution, Washington, DC.

Stuart-Macadam, Patricia. 1992. Porotic Hyperostosis: A New Perspective. *American Journal of Physical Anthropology* 87(1):39–47.

Sutton, Mark Q., and Karl J. Reinhard. 1995. Cluster Analysis of the Coprolites from Antelope House: Implications for Anasazi Diet and Cuisine. *Journal of Archaeological Science* 22(6):741–750.

Taylor, Mark J. 1985. The Paleopathology of a Southern Sinagua Population from Oak Creek Pueblo, Arizona. In *Health and Disease in the Prehistoric Southwest*, Anthropological Research Papers No. 34, edited by Charles F. Merbs and Robert J. Miller, pp. 115–118. Arizona State University, Tempe.

Temple, Daniel H. 2019. Bioarchaeological Evidence for Adaptive Plasticity and Constraint: Exploring Life-History Trade-Offs in the Human Past. *Evolutionary Anthropology: Issues, News, and Reviews* 28(1):34–46.

Thayer, Zaneta M., Julienne Rutherford, and Christopher W. Kuzawa. 2020. The Maternal Nutritional Buffering Model: An Evolutionary Framework for Pregnancy Nutritional Intervention. *Evolution, Medicine, and Public Health* 2020(1):14–27.

Trigg, Heather B., Richard I. Ford, John G. Moore, and Louise D. Jessop. 2000. Coprolite Evidence for Prehistoric Foodstuffs, Condiments, and Medicines. In *Eating on the Wild Side: The Pharmacologic, Ecologic, and Social Implications of Using Noncultigens*, edited by Nina L. Etkin, pp. 210–223. University of Arizona Press, Tucson.

Truesdale, James A. 1993. *Archeological Investigations at Two Sites in Dinosaur National Monument: 42UN1724 and 5MF2645: Data Recovery at Juniper Ledge Shelter (42UN1724)*. Selections from the Division of Cultural Resources No. 4. Submitted to US Department of the Interior National Park Service, Washington, DC.

Ubelaker, Douglas H. 1992. Porotic Hyperostosis in Prehistoric Ecuador. In *Diet, Demography, and Disease: Changing Perspectives on Anemia*, edited by Patricia Stuart-Macadam and Susan Kent, pp. 201–217. Aldine de Gruyter, Hawthorne, New York.

US Department of Agriculture. 2021. Game Meat, Rabbit, Wild, Cooked, Stewed. *FoodData Central Food Details*. Electronic document, https://fdc.nal.usda.gov/fdcapp.html#/fooddetails/174348/nutrients, accessed November 7, 2024.

Varien, Mark D. 2010. Depopulation of the Northern San Juan Region: Historical Review and Archaeological Context. In *Leaving Mesa Verde: Peril and Change in the Thirteenth-Century Southwest*, edited by Timothy A. Kohler, Mark D. Varien, and Aaron M. Wright, pp. 1–33. University of Arizona Press, Tucson.

https://doi.org/10.1017/aaq.2024.61 Published online by Cambridge University Press

Walker, Phillip L. 1985. Anemia among Prehistoric Indians of the American Southwest. In *Health and Disease in the Prehistoric Southwest*, Anthropological Research Papers No. 34, edited by Charles F. Merbs and Robert J. Miller, pp. 139–164. Arizona State University, Tempe.

Walker, Phillip L. 1986. Porotic Hyperostosis in a Marine-Dependent California Indian Population. *American Journal of Physical Anthropology* 69(3):345–354.

Walker, Phillip L., Rhonda R. Bathurst, Rebecca Richman, Thor Gjerdrum, and Valerie A. Andrushko. 2009. The Causes of Porotic Hyperostosis and Cribra Orbitalia: A Reappraisal of the Iron-Deficiency-Anemia Hypothesis. *American Journal of Physical Anthropology* 139(2):109–125.

Wapler, Ulrike, Eric Crubezy, and Michael Schultz. 2004. Is Cribra Orbitalia Synonymous with Anemia? Analysis and Interpretation of Cranial Pathology in Sudan. *American Journal of Physical Anthropology* 123(4):333–339.

Watson, James T. 2008. Prehistoric Dental Disease and the Dietary Shift from Cactus to Cultigens in Northwest Mexico. *International Journal of Osteoarchaeology* 18(2):202–212.

Williams-Dean, Glenna, and Vaughn M. Bryant Jr. 1975. Pollen Analysis of Human Coprolites from Antelope House. *Kiva* 41(1):97–111.

Windes, Thomas C., and Dabney Ford. 1996. The Chaco Wood Project: The Chronometric Reappraisal of Pueblo Bonito. *American Antiquity* 61(2):295–310.

Woosley, Anne I. 1980. Agricultural Diversity in the Prehistoric Southwest. *Kiva* 45(4):317–335.

World Health Organization. 2019a. Anemia. Electronic document, https://www.who.int/health-topics/anaemia#tab=tab_1, accessed April 4, 2020.

World Health Organization. 2019b. Pneumonia Fact Sheet. Electronic document, https://www.who.int/en/news-room/fact-sheets/detail/pneumonia, accessed April 4, 2020.

Zaino, Diane E., and Edward C. Zaino. 1975. Cribra Orbitalia in the Aborigines of Hawaii and Australia. *American Journal of Physical Anthropology* 42(1):91–93.

Zaino, Edward C. 1967. Symmetrical Osteoporosis, a Sign of Severe Anemia in the Prehistoric Pueblo Indians of the Southwest. In *Miscellaneous Papers in Paleopathology*, Vol. 1, Technical Series No. 7, edited by William D. Wade, pp. 40–47. Museum of Northern Arizona, Flagstaff.

Zucker, Stanley, Samuel Friedman, and Rita M. Lysik. 1974. Bone Marrow Erythropoiesis in the Anemia of Infection, Inflammation, and Malignancy. *Journal of Clinical Investigation* 53(4):1132–1138.

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